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Gastroscopy — Role in Private Practice

(Review of 100 Cases)

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THE purpose of this paper is to review the findings in 100 gastro-intestinal cases seen in private practice in which gastroscopic examinations were carried out. Other series have been published by workers taking their material from large hospital clinics. Believing that it might be interesting to see just what role gastroscopy plays in everyday private practice those examinations which were done in University hospitals, etc., are purposely excluded from this report. The statistical tabulation of gastroscopic diagnosis will be shown and each group of gastric lesions seen will be briefly discussed. Case histories of some of the more interesting conditions will be presented. The following is a tabulation of the frequency of various conditions seen:

Normal stomach	27
Hypertrophic gastritis	29
Superficial gastritis	18
Atrophic gastritis	7
Benign ulcer	7
Carcinoma	6
Sarcoma	1
Benign polyps	2
Post operative stomach	3
Total	100

[These figures show a remarkable similarity to those in Schindler's series of 1255 cases. The great bulk of his cases represent those done in large free clinics.]

HYPERTROPHIC GASTRITIS

This condition was found most frequently in this series of 100 cases. The gastroscopic picture here shows a dull swollen mucous membrane with granular nodules, and very frequently erosions involving the mucous membrane. Microscopic sections show a proliferation of the mucous membrane and cellular infiltration of the interstitium. All of these patients complained of epigastric discomfort. Some described a burning pain, others spoke of a dull heavy type of pain. Whereas some felt better after eating, others felt better on an empty stomach. In short, there was no typical, uniform complaint such as we usually see in peptic ulcer. Gastric analysis may show a normal content of free HCl, but usually there is a decrease in free HCl roughly proportionate to the degree of chronicity. Although this condition may cause as much discomfort to the patient as does an ulcer it may be associated with other disturbances of the gastro-intestinal tract, and the gastroscopist must be cautious about ascribing every symptom to minor changes in the mucous membrane. That these changes in the mucous membrane may also act as a precursor to carcinoma is suggested by the fact that two cases which underwent surgery at the Mayo Clinic[1] were shown to have a benign hypertrophic condition at biopsy, and within four and fifteen months respectively each again underwent surgery and carcinoma was found in the previously benign hypertrophic gastritis. One of our cases showed a large filling defect by x-ray. The radiologist referred the case for gastroscopy where the lesion was seen and believed to be a benign hypertrophic condition involving the cardia. The patient has been on symptomatic treatment for three months during which time he has felt well, retained his ample weight, and feels generally improved. The radiologist recently examined him again and stated that the lesion was smaller; this was also shown at the second gastroscopic examination. Though no biopsy was possible in this case, his clinical course seems to substantiate the gastroscopic diagnosis[2]. Schindler has recently presented seven cases of proved hypertrophic gastritis simulating tumor. A high percentage of gastro-intestinal complaints following gastric surgery have been shown to be due to this condition which were formerly thought to be due to recurrent ulcer. Although there is no specific treatment for hypertrophic gastritis considerable relief can be brought about by intelligent management.

SUPERFICIAL GASTRITIS

This condition was found in 18% of this series of cases. Though superficial gastritis comes under the heading of the chronic group it has many features which resemble the acute forms of gastritis. It is the most responsive to treatment and in most cases goes on to complete recovery. Gastroscopically we see a very swollen mucosa which is usually hyperemic in contrast to the hypertrophic type. Shallow ulcerations and hemorrhages are usually seen. The etiology of this condition is no more certain than is that of peptic ulcer, but it seems that chronic irritations, foci of infection, and an inherent tendency are the most important factors. One of our cases was a man 35 years of

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FIG 1

FIG 2

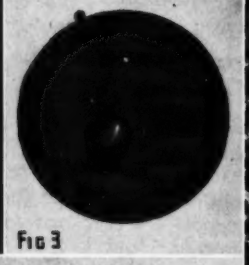

FIG 3

FIG 4

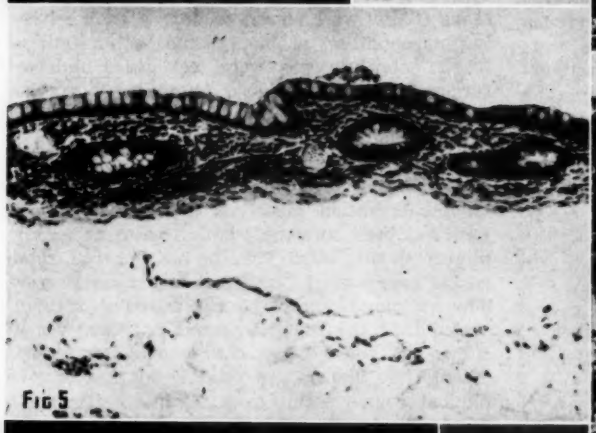

FIG 5

FIG 6

FIG 7

FIG 8

FIG 9

age, from a ranch in West Texas. He had been vomiting daily for the past six weeks, and complained of midepigastric distress together with a weight loss of 15 pounds. Physical examination disclosed no abnormal findings other than a very severe pyorrhea. X-ray examination showed no filling defects in the stomach or bulb. On 10-7-39 gastroscopic examination showed an extreme edema of the entire mucosa with ulcerations and hemorrhagic erosions. He was put to bed for ten days on an ulcer-like regime and became symptomless in two days. Two weeks later he had his teeth removed. He was seen again on 11-20-39. At this time he showed a weight gain of twelve pounds in spite of his dental ordeal and had no complaints. Gastroscopy at that time showed a very different picture from before. There were no erosions or hemorrhages and the swelling had subsided so that the mucous membrane was almost normal in appearance. Schindler[3] states that without adequate medical care about 20% of the cases of superficial gastritis go on to the atrophic type.

ATROPHIC GASTRITIS

This condition was found in 7% of this series. It presents a very definite gastroscopic picture, and is an abnormality which causes considerable distress to the patient. Its relation to other constitutional diseases makes it a most interesting condition to study. The gastroscopic picture presents a thin, grayish mucous membrane, the thinning of which makes it possible to visualize the blood vessels lying in the submucosa. These atrophic changes may be present throughout the entire stomach but are more usually seen near the cardiac end. Very frequently erosions or hemorrhagic areas are also seen. The microscopic picture shows a thinning of the mucous membrane with a cellular metaplasia resembling goblet cells, and extensive infiltration or round cells. The patient suffering from this condition complains of various types of epigastric discomfort, and characteristically complains of general constitutional symptoms as well. Thus[4] in a study of 41 cases of uncomplicated atrophic gastritis it was found that anorexia, nausea, pain, loss of weight and discomfort after eating were the principal digestive complaints, and in two-thirds of these cases complaints of weakness and fatigue appeared. This is in sharp contrast to findings in other benign gastro-intestinal disorders. Several theories have been postulated to explain this fact. 1. The atrophic mucosa may produce a toxin which is absorbed by the blood. 2. Some unknown substance present in the normal mucosa which normally prevents the development of general weakness, etc., may be absent in the atrophic mucosa. 3. The utilization of food, especially protein, may be disturbed in the atrophic stomach. 4. The complete absence of gastric acid may inhibit the functions of other organs, especially the pancreas. Gastric analysis indicates that only about one-third of the cases have a complete achlorhydria so it must be assumed that typical symptoms are more the result of anatomic changes in the gastric mucosa than as a result of the achlorhydria. The relation which atrophic gastritis bears to other diseases is an interesting study. All gastroscopists agree that carcinoma of the stomach is more likely to develop from the soil of an atrophic gastritis. Hurst[5] has shown that chronic gastritis was present in 75% of gastric carcinoma and that this condition was present for years preceding the development of carcinoma. One of my cases had been treated for years for pernicious anemia. She did have an achlorhydria and a high color index, although blood study by a competent hematologist did not show typical morphological changes for pernicious anemia. On gastroscopic examination a cancer in the antrum of the stomach was found early enough to allow resection. It is most likely, considering the time element, that she had a preceding atrophic gastritis from which the cancer developed. There is general agreement amongst pathologists, gastroscopists, and clinical gastroenterologists that this relation between atrophic gastritis and carcinoma exists. It is for this reason that Eusterman of the Mayo clinic amongst others emphasizes the importance of gastroscopic and x-ray follow up in these cases. Pernicious anemia also bears an interesting relation to atrophic gastritis and it is possible that the direct observation of the gastric mucosa in this disease may assist in throwing new light on its pathology.

Much can be done to alleviate the distress caused by atrophic gastritis. Only one of this group failed to show very definite subjective improvement on a regimen which consisted of dietary and medical management. One of these patients had previously been through a very extensive diagnostic work up in an attempt to find a malignancy which his appearance and clinical story suggested. On gastroscopic examination an almost complete atrophy was seen together with mucosal erosions. On simple management he gained 40 pounds over a year's time and was symptom-free for long intervals, having only relatively mild periodic recurrences of distress. Frequent gastroscopic examinations, however, showed no improvement of the atrophic condition of the mucous membrane. Very recently[6] desiccated hog's stomach (ventriculin) in massive dosage has been shown to bring about an actual disappearance of the atrophic changes. The obvious suggestion is that atrophic gastritis may be due to a deficiency of some substance which is present in desiccated hog's stomach extract. If

Fig. 1.—Showing one polypoid growth near the cardia.
Fig. 2.—Note the greyish, swollen appearance of the mucous membrane, indicative of an atrophic gastritis, and the presence of 3 polyps near the pyloric orifice.
Fig. 3.—Showing the large polyp seen in roentgenogram (Fig. 1). Mucous membrane again shows a picture of atrophic gastritis.
Fig. 4.—Normal stomach mucosa.
Fig. 5.—Atrophic gastritis.
Fig. 6.—Chronic gastritis.
Fig. 7.—Author's case of lymphosarcoma of stomach—x-ray view.
Fig. 8.—Author's case of lymphosarcoma of stomach—sketch of gastroscopic view of mass in antrum of stomach.
Fig. 9.—Author's case of lymphosarcoma of stomach.

these findings are further substantiated it will be a most important discovery not only as a treatment for the disease itself, but also as a prophylactic for potential gastric carcinoma and possibly pernicious anemia.

BENIGN ULCER

In this series we find seven cases of true gastric ulcer. In the diagnosis of this condition we have a splendid example of the value of gastroscopy as a complement to x-ray and vice versa. In a controlled series of cases it was found that both methods had their advantages and were able to demonstrate the lesion where the other failed to do so. It is known that lesions in or near the cardia are often difficult to demonstrate radiologically. In these cases gastroscopy may be of great aid. Furthermore, in ulcers which have a very shallow crater the displacement of barium may be so slight as to not allow its visualization. Schindler has stated that complete epithelialization of ulcer can be determined only by gastroscopy and that this completion may require as long as two months after refined x-ray compression has failed to visualize any abnormality. This fact was demonstrated in one of the above cases. A compensation case was brought to court and the decision rested on whether a gastro-intestinal lesion was present. X-ray examination failed to show any abnormality, but gastroscopy showed an area of intense swelling surrounding a healing gastric ulcer. It is in the differentiation of benign and malignant lesions, however, that gastroscopy probably finds its greatest usefulness in connection with gastric ulcers. The gastroscopic picture of each is quite distinctive. The benign ulcer presents a punched out appearance with clean-cut margins and a smooth grayish floor. In contrast to this the malignant lesions usually show a nodular formation with ill defined margins, and frequently the presence of blood coagula. In addition the process may involve the surrounding tissue. One of the above cases was spared a gastric resection because of the demonstration of an antral lesion's being benign. On the other hand we have presented cases which were called benign by x-ray and malignant by gastroscopic examination and which by biopsy were shown to be early malignancy.[7]

CARCINOMA

Early diagnosis of gastric carcinoma is the goal of every physician, and any procedure which will be helpful in this respect deserves the greatest consideration, especially when we remember that 25% of all cancer deaths are due to gastric malignancy. In the opinion of the author the greatest difficulty in diagnosing early gastric carcinoma lies not so much in our inability to demonstrate the lesion by x-ray or gastroscopy, but rather in getting the patient to present himself to a physician while the lesion is still in the early stage. Admitting this difficulty we must still concede that many cases which have presented themselves to physicians have been overlooked until such time that the diagnosis was evident without the aid of any refined procedure. It is this group which challenges our diagnostic skill. The case mentioned briefly under atrophic gastritis is a good example of a missed diagnosis of an early lesion until gastroscopic examination disclosed its presence. Although this patient had been consulting with physicians for six months, no attempt was made to examine her gastro-intestinal tract because they assumed that because her blood picture resembled that found in pernicious anemia, that that condition existed and explained the symptomatology. This is a very dangerous assumption since constant slow seepage of blood from the gastro-intestinal tract may frequently give a hyperchromic type of anemia. Fortunately the correct diagnosis was established sufficiently early to allow resection.

It seems that it is an idle argument as to which is the superior for diagnosis of an early carcinoma, x-ray or gastroscopy. The statement which I feel no one will object to is that the more that we can know about any questionable lesion of the stomach, the more likely we are to handle the case to the greatest advantage of the patient. Gastroscopy then becomes of great value in checking a suspicious area which may be seen by x-ray and which may possibly be due to spasm, extrinsic pressure, etc. Ideally all gastric ulcers should be observed gastroscopically because of the characteristic picture which is afforded of benign and malignant ulcers. It has been shown that the earliest malignant changes of a benign ulcer have been recognized by gastroscopy. In this series of seven cases all were diagnosed correctly by gastroscopy and six of the seven cases had biopsy proofs. Since atrophic gastritis has been definitely shown to be the soil in which carcinoma is likely to develop, isn't it reasonable that known cases of atrophic gastritis should be observed at intervals, following much the same reasoning as in having periodic chest x-rays or people exposed to tuberculosis? Furthermore, if we are so concerned about early diagnosis of carcinoma it might be wise to include atrophic gastritis in our study of the cancer problem as a whole.

SARCOMA

Sarcoma of the stomach comprises about 1% of gastric neoplasms. Ewing classifies them as follows: 1. Spindle cell myosarcoma. 2. Miscellaneous round cell or mixed cell alveolar sarcoma. 3. Lymphosarcoma. The last mentioned constitute the largest and most important group. In the case to be mentioned the usual detailed case history will be omitted for the sake of brevity, and only the essential facts will be given. The patient, presented himself at the office with a history strongly suggesting peptic ulcer. His distress dated from the previous year, but he had no weight loss, and there were frequent remissions in his symptoms. His hbg. was 87%, R.B.C.: 4.6, free HCl 18° and total acid 43°. All other laboratory findings were within normal limits. The radiologist reported the presence of a duodenal ulcer. On ambula-

tory management he did not do well, and because of pain which persisted even during a strict hospital regimen he was gastroscoped one month following x-ray. Following were the findings: "The pyloric end of the stomach was well seen. The antrum was completely involved by a grayish irregular growth. Some infiltration involved the angulus. The upper parts of the stomach showed edema and hyperemia, but no tumor infiltration. Impression: Malignant growth of the antrum, possible lymphosarcoma." The patient was again x-rayed at which time the conclusion was that a carcinoma was present in the antrum. At surgery the lower third of the stomach was found to be involved in a tumorous mass extending down into the duodenum. Biopsy report showed the tumor to be a dark cell type lymphosarcoma. Because of the involvement of the duodenum a resection was not attempted, but a posterior gastro enterostomy was done and was followed by 15 treatments of 150% erythema dose of deep x-ray therapy. The patient showed great improvement for a while and went back to work. Three months later he began to decline and was given more deep therapy.

The author had studied one other case of gastric lymphosarcoma by gastroscopy with Schindler, and because of the remarkable similarity of the gastroscopic picture it was possible to diagnose the lesion correctly. This case illustrates the value of gastroscopy in diagnosis of obscure lesions, and also the great accuracy which it makes possible. Total extirpation of the lesion is the desired goal in treatment of lymphosarcoma followed by radiation therapy.

BENIGN TUMORS

Benign tumors are relatively rare and constitute about 1.3% of all gastric tumors. They occur in comparison to malignant tumors in ratio of 1:200. They are of interest because of their tendency to malignant degeneration. In this series of 100 cases, two cases of multiple benign polyps were found. In a review of 34,670 autopsies at the University of Minnesota we found only 32 cases of this condition. Figures 1, 2 and 3 show how clearly the polyps were demonstrated in contrast to x-ray.

CONCLUSIONS

100 gastro-intestinal cases which were examined by gastroscopy, and which were seen in private practice have been reviewed and statistics concerning the frequency of various conditions have been tabulated. Each group of cases has been discussed and certain cases have been individually reviewed. It seems quite clear that gastroscopy is of great diagnostic value in general private practice, and this review seems to substantiate the statement made by Henning[3] of Germany that no diagnostic work up of the stomach was complete without gastroscopic examination. Eusterman[8] recently stated: "It follows that gastroscopy should be looked upon as an indispensable diagnostic procedure in daily medical practice. Routine gastroscopic examination is advised in every case in which a complaint of consistent dyspepsia is made and in which roentgenologic findings are negative; such an examination often discloses the presence of inflammatory mucosal lesions which, until recently, were unrecognized and untreated in this country."

Mills Bldg.

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Incidence of Agglutinins for Typhoid, Paratyphoid and Brucella Abortus

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GREGORY and Atkinson[1] have pointed out that "for the correct interpretation of the Widal test it is desirable to have information regarding the natural level of agglutinins among a random sample of the population in the particular area in which the test is being made." Topley and Wilson[2], as well as others, have pointed out that the sera of persons who have no history of enteric infection or of immunization may contain agglutinins for typhoid or paratyphoid—often in titers which might be considered as diagnostic.

* From the Arizona State Laboratory, Tucson, Arizona.

In order to determine to what extent agglutinins might occur in the sera of residents of Arizona, all blood specimens which have been submitted to this laboratory have been examined for the presence of agglutinins for typhoid, paratyphoid A and B, and Brucella abortus. These specimens, in general, have been submitted from the southern part of Arizona. The majority have been submitted for serological tests for syphilis, and presumably in these cases, there was no indication of enteric or Brucella infection at the time the specimen was taken. These specimens represent every race, oc-

cupation, social and economic level found in the southern portion of the state. We feel, therefore, that 10,000 specimens represent a fair sampling of the area.

EXPERIMENTAL METHODS

The rapid slide method of Welch and Stuart[3] was employed for the detection of typhoid and paratyphoid agglutinins. The method of Huddleson[4] was employed for the detection of Brucella agglutinins. The antigens were manufactured by Lederle Laboratories, except that a portion of the brucella antigen was perpared by Miss Barbara Wilson, technician, Co-operative Bang Eredication Project from a culture supplied by the Bureau of Animal Industry. All tests were performed upon unheated sera, and were carried out on a glass plate, as suggested by Huddleson[4], rather than on slides, as suggested by Welch and Stuart[3]. Although it has not been possible to compare the tube and plate methods in every case, comparisons which we have made have shown very satisfactory agreement.

RESULTS

A total of 10,000 sera were examined; 9515 did not agglutinate any of the antigens and 485 agglutinated one or more of the antigens in a dilution of 1:20 or greater. Due to the number of possible combinations which might be obtained through the use of five antigens and five serum dilutions, the results cannot be given in detail.

The maximum agglutination titers (i.e., a serum agglutinating in dilution of 1:40, but not 1:80, is listed as 1:40, and not in 1:20 and 1:40) observed are given in Table I. Although higher dilutions have been made in some cases, they are not included. The first column under each dilution contains the number of sera agglutinating the particular antigen in a maximum serum dilution as indicated at the top of the column. The second column (in parenthesis) is the number of sera, agglutinating in that particular dilution, in which there is a definite history of infection or immunization. The third column gives the percentage of these specimens in which there is a positive history of immunization or infection.

The results of the Brucella agglutinations are expressed, for uniformity, on the same basis as the other agglutination tests. Actually, according to the method of Huddleson, they should be reported as 1:25, 1:50, 1:100, etc.

Table II gives the number of sera agglutinating the various antigens in dilutions of 1:20 or greater. Although it is not possible to give all of the combinations, those which appear represent the majority of the reactions observed.

TABLE II

Combinations of antigens agglutinated in dilutions of 1:20 or greater—

Antigens	Number of Sera	Number of Known Infections of Immunizations	Percentages of Known Infections of Immunizations
Typhoid H	48	43	90%
Typhoid O	63	42	67%
Paratyphoid A	27	15	55%
Paratyphoid B	10	8	80%
Brucella Abortus	27	17	63%
Typhoid H. and O.	90	72	80%
Typhoid H, O, Paratyphoid A & B	147	140	95%
Typhoid H, O, Paratyphoid A, B, & Brucella abortus	3	0	0
Typhoid H, Paratyphoid A & B	13	9	69%
Paratyphoid A & B	6	0	0
Typhoid H. and Paratyphoid A	6	4	66%

In all cases it has not been possible to get complete histories of infection or immunization. Fairly complete records were secured from students at the University of Arizona. In September, 1938, 1042 blood specimens were submitted by the University Health Service for new students. The results are:

Number of Specimens	1042
Number agglutinating one or more antigen in a dilution of 1:20, or higher	42
Number of known immunizations or infections	32
Percentage of agglutinations	4%
Percentage of agglutinations, "Positive History"	3%

If these University students are used as a basis of comparison, the incidence of agglutinins among persons who have no history of immunization or infection is approximately 1%.

Table III gives, in detail, the agglutination reactions of these students who had no history of immunization.

TABLE III

Agglutination reactions of students with no history of infection or immunization—

Student	Typhoid H	Typhoid O	Paratyphoid A	Paratyphoid B	Brucella abortus
M. A.	——	1:40	——	——	——
C. P.	——	1:40	——	——	——
A. K.	——	1:40	——	——	——
M. B.	1:320	1:320	1:320	1:320	——
M. P.	1:320	1:320	1:320	1:320	——
B. S.	——	——	1:40	——	——
I. P.	1:40	——	——	——	——
L. P.	1:40	1.40	——	——	——
N. G.	1:40	1:80	——	——	——
W. H.	——	1:40	——	——	——

In a group of 154 students at the Arizona School for the Deaf and Blind, 145 did not agglutinate any antigen. Of the nine who did agglutinate one or more antigens (5.8%), five had histories of immunization or infection. In the group of four who

TABLE I

Maximum agglutinating titers

(Figures in parenthesis indicate number of known infections or immunizations, percentages refer to known "positive histories".)

Antigen		1:20			1:40			1:80			1:160			1:320	
Typhoid H	6	(4)	66%	198	(184)	93%	68	(48)	71%	29	(18)	62%	33	(25)	76%
Typhoid O	8	(5)	62%	172	(156)	91%	43	(27)	63%	28	(21)	58%	38	(28)	74%
Paratyphoid A	7	(3)	43%	140	(127)	91%	40	(30)	75%	15	(9)	60%	17	(12)	71%
Paratyphoid B	5	(3)	60%	117	(115)	88%	37	(32)	87%	5	(5)	100%	20	(7)	35%
Brucella abortus	4	(4)	100%	10	(4)	40%	12	(6)	50%	4	(1)	25%	12	(9)	75%

had no history (2.6%), one agglutinated Typhoid H, 1:80; one, Typhoid H and Typhoid O, 1:40; one Typhoid H, 1:80 and one Typhoid O, 1:160.

In a group of 132 immunized children at the Pima County Preventorium, 19 showed some agglutination with one or more of the antigens.

DISCUSSION

The preceding tables have shown that, in general, the incidence of agglutinins among persons who have no history of infection or immunization is quite low. In a series of 10,000 sera, only 485 (4.85%) showed any agglutination. Since a large proportion of these had histories of immunization or infection, the incidence of agglutinins among persons with no history is lower than that reported for Alabama by Havens[5], Oregon by Sears*, and those given by Topley and Wilson[2].

Twenty-seven sera (0.27%) agglutinated Brucella abortus; 17 (63%) had been clinically diagnosed, out of the ten with no history, one was a butcher, one a dairyman, and a large percentage of the remainder were Indians. This percentage is lower than that reported by Gersh and Murgrage[6] who found that 1.2% of a series of 5,000 sera submitted for a Wasserman test gave agglutination with Brucella antigen.

Shaughnessey and Grubb[7] have reported from three to nine per cent positive brucella agglutination tests in tubercular patients who gave negative skin tests for brucella infections. Although we have no records as to the number of tuberculars in our series, we have reason to believe that they are fairly great. Because of the lack of this information, it is not possible to compare our results directly with those of Shaughnessey and Grubb. It seems quite possible, however, that some of the agglutinations reported for persons with no history of immunization or infection, may be non-specific and may be due to tuberculosis or other infections.

During the course of this investigation, we have had the opportunity of examining specimens from known cases of infections (or immunizations) over a fairly long period These results have shown that the duration of agglutinins following immunization or infection is extremely variable. In some cases, they may persist in fairly high titers for years; in other cases they may disappear within a few months.

Anamnestic reactions have been observed frequently. Immunized persons, whose sera had previously not agglutinated any antigens, have subsequently shown agglutinins in fairly high titers in illness other than enteric ifections. The agglutinin titer usually dropped rapidly with recovery.

When this investigation was begun, we hoped that we might be able to establish some arbitrary titer which might be considered as diagnostic for enteric infections. Such a diagnostic titer has been widely discussed, and excellent accounts are given by Havens[5] Topley and Wilson[2], and Hac, Flynn and Perry[8].

*Communication from Dr. H. J. Sears.

In cases of enteric infections which have been clinically and bacteriologically diagnosed, we have noted wide variations in the production of agglutinins. In some cases, only "H" or "O" agglutinins are produced. We have noted a relatively high incidence of "O" agglutinins among immunized individuals. We have noted "H", "O", and "H and O" agglutinins in rather high titers as anamnestic reactions in immunized persons, and also as presumably non-specific reactions in persons with no history of infection or immunization.

Since less than five per cent of the sera examined gave any agglutination, and since a large percentage of these gave histories of immunization or of infection, it seems that the incidence of natural agglutinins is quite low. Consequently, we feel that (under conditions in southern Arizona) agglutination with any antigen at any titer may be considered as significant when there is no history of immunization, and when clinical symptoms suggest enteric or brucella infection. In cases which are not clinically typical, anamnestic or non-specific reactions may be eliminated only by demonstrating a rise in tier, by means of a series of specimens, or by isolation of the causative organism.

CONCLUSION

In a series of 10,000 blood specimens, 485 (4.85%) agglutinated one or more of the following antigens: Typhoid H, Typhoid O, Paratyphoid A, Paratyphoid B, Brucella abortus.

The incidence of agglutinins for typhoid or paratyphoid in persons with no history of immunization or infection is estimated at 1%.

In this series of 10,000 specimens, 27 (0.27%) contained agglutinins for Brucella abortus. Seventeen of these had been clinically diagnosed.

The occurrence of anamnestic and non-specific reactions is discussed.

It is suggested that, under Arizona conditions, agglutination with any antigen, in any titer, may be considered significant when clinical symptoms are suggestions of eteric or Brucella infection, and when there is no history of immunization.

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Carcinoma of the Anus, Rectum and Recto-Sigmoid

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CANCER of the rectum takes a toll of about 35,000 lives in the United States each year. Although it occurs more often between the ages of 50 and 70, it also affects younger people, cases having been reported as early as four years of age. In the group of almost 500 patients examined, I found 14 to be below 30 years of age, and 36 to be between 30 and 40, a total of 50, or 10%, occurring in the span of life in which cancer is the most fatal.

As is customary in all papers dealing with this subject, I wish to make the plea for the education of the patient and the physician in the early recognition of this condition, that these cases may be treated when there is still the chance for cure. The only way that cancer of the rectum and recto-sigmoid may be eradicated, in the light of our present knowledge, will be through periodic examinations, and the destruction by the electric cautery of every adenoma or polyp, no matter how small or innocent in appearance it may be. When the classical symptoms of bowel frequency, with blood, pus and mucus, loss of weight and anemia, abdominal pain and distention, are noted by the patient, the disease is usually well advanced. Bleeding, which is the most important finding in any rectal condition, may come from hemorrhoids, fissure, polyp, parasitic infection, ulcerative proctitis or colitis. The examination with the finger and the proctoscope should never be omitted, even though parasites are found in the stool, or a fissure or hemorrhoids are demonstrated by anoscopic examination. The barium enema should be withheld until after the proper sigmoidoscopic examination has been made. A biopsy of suspected tissue should be taken in all cases. The old idea that the removal of tissue might spread cancer cells is unfounded. Grading of the specimen by the pathologist is only one finding which must be correlated with all the others. It is generally accepted that the higher the grade, the more malignant, and the less chance of cure, and also the more sensitive to irradiation. In primary cancer, the mucosa is always involved. Cancer grows either into the lumen of the bowel, as in the papillary and polypoid types, or into the wall of the bowel, as in the ulcerative types. The growth is practically always hard, distinctly outlined, rough, and ordinarily bleeds easily. It may entirely surround the bowel as in the annular type, or may be restricted to a local area of the bowel surface. Primary cancer must be distinguished from ulcerative lesions as: infections from parasites, ulcerative colitis, and lympho-granuloma inguinale; from secondary involvement by implants from malignant lesions higher in the abdominal cavity as: cancer of the stomach, pancreas, or ovary; from extension from cancer of the prostate; from proctitis due to the effect of radium and x-ray in the treatment of cancer of the uterus and cervix; from inflammatory nodules; endometrioma; and benign growths.

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TREATMENT

In the consideration of the treatment, we must keep in mind that the fundamental reason for treatment is the cure of the disease, or the prolongation of life, with the least possible temporary or permanent impairment of bodily function of the patient. The selection of the type of treatment depends upon the physical condition of the patient, the location and characteristics of the tumor, and the presence or absence of metastases. The surgeon who attempts to use one procedure in all cases will either have a high mortality rate, a high recurrence rate, or a low operability rate. The procedure selected must be suited to each individual patient after careful evaluation of clinical and pathological findings. The most we can promise any patient with cancer of the rectum, is that there is a reasonable chance that life will be continued with the minimum amount of disability and discomfort.

Metastases occur by cancer cells being carried through the venous and lymphatic circulations. We are more concerned with the lymphatic circulation when we select the method of treatment. If we do not remove the growth and the lypmphatic structures which are above it, we are very prone to have a continuation of the disease. Considering the most complete removal of all lymphatic structures as a criterion, the abdominoperineal resection offers the best chance for cure. This procedure may be done in either one or two-stages, depending upon the conditio nof the patient and the experience of the operator. The two-stage is the safest, but it entails more surgery and more hospitalization.

The procedure which offers the next best chance for cure, because it removes somewhat less of the gland bearing tissue than the abdominoperineal, is the colostomy and perineal resection. This method is excellent in poor risks with low tumors. I feel that any growth, above which the finger can not reach, is not suitable for this operation and that some form of the abdominal type, the condition of the patient permitting, should be used. The mortality rate in ordinary cases is lower, but the recurrence rate is supposed to be somewhat higher than in the abdominoperineal operation.

The third preference would be the colostomy and perineal excision, which means that the bowel is severed below the peritoneal reflexsion. This may

also be performed without colostomy, and is not to be considered rightly as a radical procedure but more as a local resection. Recurrence rate is much higher than in the other procedures. Local excision should be classed only as a palliative procedure.

It is now the consensus that even in the presence of moderate glandular and liver metastases, the tumor itself should be removed, in order that the patient be made more comfortable, and the spread of the disease be retarded.

In treating skin cancers, or the epidermoid type in the anus or lower rectum, I favor the use of radium, deep x-ray therapy, and surgical excision. Our results with surgery alone have not been gratifying. A temporary or permanent colostomy is usually necessary. In the adenocarcinomas, we feel that x-ray and radium therapy are to be used only in those cases deemed inoperable, either from the condition of the tumor, or the condition of the patient. We are all aware that some cases are reported as having been cured by these methods, but we should not consider them justifiable in the average operable case.

CARE OF PATIENT

Whatever surgical method we pursue, or technique we use, we must pay strict attention to preoperative and post-operative care. The patient should not be operated, ordinarily, with less than a week's hospital care; the bowel should be decompressed by oil and saline laxatives and enemas. If the blood picture is low, transfusions are indicated; the function of the kidneys and heart and blood vessels must be determined by the proper examinations. The strength of the patient must be kept up by high caloric, high crabohydrate and low residue diet.

Post-operative care must be meticulous in that the patient must be watched for signs of shock and hemorrhage. The blood pressure should be taken at frequent intervals, and if below normal, should be stimulated by the use of ephedrine in suitable doses. Oxygen should be used, especially if there is a rapid pulse, and a blood transfusion should be given immediately following any radical procedure. Intestinal drainage should be instituted by the nasal tube when there is the slightest upper abdominal distension or nausea. The bowel should be opened in 24 to 48 hours, and if there are no contra-indications, fluid and food should be started early. The fluid balance is kept up by the intravenous use of glucose.

Complications to anticipate are: peritonitis, gastric dilatation, paralytic ileus, mechanical obstruction, embolism, pneumonia, and post-operative hemorrhage.

SUMMARY

1. Only by proper and frequent examinations will cancer of this region be controlled.

2. The choice of procedure must be chosen for each individual patient, depending upon the clinical and laboratory findings.

3. We must be conservative regarding life, but boldly aggressive in attempting the cure of cancer.

1930 Wilshire Blvd.

Tuberculosis of the Spine

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TUBERCULOSIS of the spine is so important a condition, because of its protracted course and serious complications, that a review of the outstanding facts concerning the disease is not out of place.

Vertebral tuberculosis is essentially a disease of childhood; often it occurs where the family history is one of tuberculosis.

The part of the spine most frequently involved is the lower thoracic and upper lumbar spine; but other regions are susceptible. More than one lesion may be present. The body of the vertebra is usually involved, but the infection may originate in any part. The marrow is infected and destroyed; with growth of the tubercle, adjacent bony lamellae are rarefied and disappear. Central caseation occurs, with collapse of the weight—bearing structures; an angular deformity, the gibbus, results. Caseous material escapes about the vertebra to form a "cold" abscess; it may remain localized by the spinal ligaments, or disseminate along various fascial planes, to make its appearance at considerable distance from its origin. Pressure on the spinal cord may result from abscess, angulation of the bony canal, and from ingrowing granulations, producing disturbances of reflexes, sensation, and motor function.

Bony fusion is nature's response to limit the infection—this may occur between the posterior articular parts of the vertebrae, but usually about the involved bodies. In adults, fibrous walling off of the involved area often occurs. Absorption of tuberculous debris with later calcification and new-bone formation occurs.

Local symptoms may be elicited before definite destruction appears in the x-ray. Pain, muscle spasm, night cries, spasticity on walking, deformity and abscess formation—one or more may be the clew. The x-ray confirms the clinical diagnosis, shows the extent of the disease, presence of abscess, and any evidence of healing that may be

"Statistics On The Fusion Operation In Tuberculosis Of The Spine", 1937-38 Seminar Notes of the Department of Orthopedic Surgery, The State University of Iowa, by permission of Dr. Arthur Steindler.

present. As in so many orthopedic conditions, an early diagnosis is important for the most effective treatment. Of the laboratory procedures the intra dermal tuberculin test is most important.

The treatment of this condition is prolonged and often extensive. The usual anti-tubercular measures, such as good diet, improved hygiene, fresh air and sunlight, are highly essential. The object of local treatment is to secure immobilization of the affected region of the spine and so to permit nature to heal the condition. The details of treatment depend partly on the conditions of each case —the presence of gibbus, abscess, fistula, or paralysis. In an uncomplicated case, complete recumbency is instituted from the start; the patient is placed on a plaster bed or on a curved frame, and restraints are used when necessary. This stage of treatment lasts from 1½ months to 2 years, when the patient may be permitted up, provided certain criteria are met: (1) freedom from pain and tenderness locally, (2) good general condition, (3) no increase in kyphosis, (4) a normal temperature, (5) gaining of weight, and (6) healing seen in the x-ray.

Spinal tuberculosis is a slow and extremely chronic disease, and the treatment must not be hurried, to be effective and reasonably certain. Each patient requires careful supervision, lest a recrudescence occur. It is better to err in prolonging the treatment, rather than by default of precautions. Consolidation of the collapsed bodies may take 3 or 4 years; the x-ray is the important deciding factor. Gradually support is removed—first for short period each day, later for longer periods. The patient is watched for return of pain or increase in the degree of deformity.

Traction is often used in disease of the cervical and upper thoracic spine; during the recumbent period this is applied to the head by halter and weights. Round shoulders or short neck may be the sequelae; paralysis may occur. In the lumbar spine healing occurs quicker, and more control of deformity is possible; abscess formation may cause spasm and contracture.

Abscess formation is the most frequent complication of Pott's disease and may cause pressure symptoms or appear as a tumor. It consists of liquefied debris, with a wall of granulation tissue about; the fluid is sterile unless secondarily infected. At any stage, the abscess may absorb or calcify. It often migrates along fascial planes, blood vessels and nerves. In the retropharyngeal space, abscesses should be evacuated from the neck, to avoid rupture into the pharynx. In the thoracic region, operative interference is difficult, but fortunately rarely required; resection of a transverse process and rib gives the approach. In the lumbar region, aspiration may be necessary.

Paralysis requires absolute recumbency combined with traction or hyperextension; this may be sufficient in mild cases. But where conservative measures fail, laminectomy must be considered; fusion may be done at the same time.

Deformity is often difficult to prevent; and, once established, hard to correct. Only gradual methods can be considered as free from danger; certainly manipulations are to be condemned. The patient may be placed in hyperextension, on a Bradford frame or plaster bed.

Operative fusion of the spine is favored as the best means to control deformity, and to permit quicker and permanent healing. The various operations will produce solid fusion, thus putting at complete rest involved areas—which no external splinting can accomplish. The patient should be under observation, so that before operation he is in good condition, and is either holding his own, or improving. Operation should be avoided in the presence of secondary infection with high fever. After the operation, the spine must be immobilized at least 6 months in a recumbent position.

CASE STUDIES

An analysis of 100 attempted spinal fusions for tuberculosis of the spine* at the Crippled Children's Hospital, Iowa City, Iowa, was made in an attempt to answer several questions.

Does the operation really fuse the spine? In 87 cases in which the end result was stated, solid fusion occurred in 84 (94%). The Hibbs and Albee technique, and modifications of these, were used with equal success.

Does fusion control the deformity in tuberculosis of the spine? In 73 cases presenting deformity, this deformity either decreased or came to a standstill in 70 cases (96%).

Does fusion relieve paraplegia? Paraplegia was relieved, or its progress arrested, following fusion in 10 out of 13 cases (76%). Fusion was preceded by traction in recumbency.

Does fusion heal abscess or sinus? In 89% of 39 cases with these complications, a good effect was obtained; abscesses disappeared or calcified, and sinuses cleared up. With the healing of the tuberculous process, abscess and sinus disappear.

Does the age of the patient influence fusion? Evidently not, since each age group (by decades) seemed to have roughly equally good results. However, in the 14 patients over 40 years of age, the highest percentage of good results was obtained (93%).

Does fusion shorten the time of treatment? The average period of recumbency before operation was about 11 months; after operation about 6 months. The average period of ambulant treatment, when the patient was permitted to sit or walk with strong support, was 23 months. Since non-operative treatment usually requires about three or four years of recumbency, and about two years of ambulant care, there is a definite saving in time by fusing the spine. The saving of time may not be important; the obtaining of solid bony fusion is, since this is the only way absolute immobilization can be assured to the involved vertebrae. Non-operative treatment may result in incomplete or fibrous healing, which may subsequently break down under physical activity.

When should fusion be done? It was found that fusion attempted on a patient whose general condition was progressively getting worse always resulted disastrously. Where the general condition had remained at a constant level, or was improving, the value of the operation became decidedly greater. Cases to be fused, then, should be selected only after carful study over a long period of time —it is not enough that the patient be able to withstand a major operation. If the patient's body is not able to handle the tuberculosis infection already present, operation is contraindicated; it introduces such risks as operative shock, secondary infection, and sinus formation. It is not a last desperate therapeutic procedure.

SUMMARY

In carefully selected cases, operative fusion for tuberculosis of the spine shortens the period of treatment, controls deformity, abscess, and sinus; and offers protection against cord compression.

First National Bank Bldg.

Relapsing Fever

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RELAPSING fever is an ancient disease. Hippocrates described it as remittent malarial fever. However, no definite observations were made until 1744, when an epidemic occurred in Ireland. Since then it has occurred almost all over the world. There have been epidemics in Ireland, Scotland, England, Russia and Germany. During the World War there was quite an epidemic in Serbia. Carter Van Dyke, in 1878, described the spirillum in the blood of relapsing fever patients. This disease was not known in North America until 1844, when some cases were reported in Philadelphia, occurring in immigrants from Ireland. Later, in New York and Buffalo, epidemics occurred among people from Ireland. In 1857 Livingston reported a peculiar type of relapsing fever in Africa, which often followed the bite of a tick. It was called tick fever, and spread along the trade routes from Africa to Asia. In 1874 an eqidemic among Chinese laborers in California was reported. These previous cases in the U. S. had all been in immigrants who had contracted the disease before arriving in the U. S. There was no endemic focus in the U. S. until 1915, when Dr. Meader reported five cases coming from Bear Creek Canyon in Colorado. He believed this disease had been brought to this particular region by a band of gypsies.

In 1918 Waring[14] reported another case in a twelve year old boy, occurring in the same region. In 1922 Briggs[3] of San Francisco reported that in August, 1921, he had two patients who contracted the disease in Polaris, California. These two patients each noticed a little blood spot on the sheet, and some urticarial spots on the legs from some insect bite. Early in May, 1925, Dr. R. O. Raymond[11] of Flagstaff, Arizona, had under observation a patient that was diagnosed as relapsing fever by the Pathological Laboratory in Phoenix and confirmed by the Hygenic Laboratories in Washington, D. C. This patient obtained the infection in northern Arizona, presumably near Greer, Arizona. The first case reported in Texas was in 1927. In 1930[12] first proof of relapsing fever was found in Smith Valley, Nevada. Since that time thirty known cases have been found. In 1930 Dr. Bannister[2] of Phoenix, Arizona reported a case of relapsing fever in a man who had contracted the infection around Greer, Arizona. In 1931 Graham[7] reported four cases occurring in central Texas. These occurred in four boys who had gone exploring a cave infested by ticks. This cave, called the Blue Bug Cave, had a bad reputation because people who entered became mysteriously ill. This had been known for at least ten years, and Graham[7] was able to obtain information of about forty cases of relapsing fever coming from that locality. In 1932 Varden[13] of San Bernardino, California reported the disease in a five year old child, contracted in Big Bear Valley in California. In 1933 Legge[9] of Berkeley reported the disease in a medical entomologist who had been employed by the California State Board of Health to make a survey of certain ticks found on rodents. This man sustained a scratch on the right thumb and a raw area on his hand from a burn. While handling a freshly killed squirrel he contaminated the wounds. Eight days later he developed the disease. Blood smear from the squirrel was found positive for spirochetae.

In 1935 Gillespie[6] reported two cases of relapsing fever occurring in Texas. In his article he states that data had been recorded on 258 cases occurring in Texas in the previous five years. After the appearance of an epidemic around Gold Park, Nevada[12] in 1935, at which time eleven persons developed the illness, natives said that there had been a similar malady fifteen years before, affecting numerous persons, and several had died. In 1936, Burns[4] of Walnut Park, California reported a boy seven years old, (his own son) who contracted the disease at Big Bear Lake. In 1938 Dr. Irvine[8] of Tempe, Arizona, had a patient who contracted relapsing fever near Greer, Arizona.

ETIOLOGY

The cause of relapsing fever is a spirochete of the genus Borrelia which produces septicemia in man. There are no particular morphological differences in the different species of spirochetae, but

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on the basis of serological studies, several different species have been isolated. In America it is known as Borrelia Novyi.

MODE OF TRANSMISSION

In the southwestern part of the U. S. the disease is carried from the reservoir hosts by the ticks of the genus Ornithodoros. After the insect has bitten an infected animal, the spirochetae disappear from its alimentary tract, and in six or seven days are found in all the body tissues. This tick, once infected, will transmit the disease to its offspring, so the egg, larval, nymphal and adult forms are all infected. Probably, from clinical evidence, the least dangerous, since its presence on the human body is noticed, is the adult form, while the nymphal forms may obtain blood without causing any disturbance to man. This fact may be the reason why so few cases remember an insect bite. Feces from the insect are scratched into the wound from which the blood meal was obtained, and the spirochete enters the human body.

It[12] has been found that many animals are able to harbor the infection; notably the monkey, opossum, armadillo, calf, horse, cat, chipmunk, ground squirrel, chicken, dog, rat, and tamarack squirrel. Muhlens and Grothusen have found that children and adults may act as carriers, and that spirochetae have been found in the blood stream of symptomless patients as long as ninety-eight days after finding a positive smear. The incidence of the disease is greater among males, probably due to greater exposure, but no age group is immune. Most infections occur during the summer months, when the rodents make their appearance.

SYMPTOMS AND SIGNS

Incubation period varies from six to nine days. The patient suddenly becomes acutely ill, although there may be a prodromal period of general malaise. The onset is abrupt, with high fever. The patient may have chills and intense headache, severe aching in the back, arms and legs. Very frequently early in the disease there is nausea and vomiting. The temperature may go up to 104° or 105°. This febrile period may last from two to five days. The temperature usually gradually diminishes somewhat during these days, then suddenly drops by crisis. There is profuse sweating, and the patient feels well except for weakness. During the attack both diarrhea and constipation have been reported. The spleen may become enlarged. With remission the spleen diminishes in size. With a relapse, it increases in size and becomes tender. The afebrile period lasts from two to four, five, or six days, during which time the patient feels quite well. Another relapse may occur with sudden onset as before. In medical books Cecil[5] and Meakins[10] report that many cases terminate with one relapse. A few go through three relapses. More than that is rare. However, in the literature reviewed, this does does not seem to be true, since patients are reported as having as many as ten relapses. The patient reported by Waring[14] was not treated specifically, and had five bouts of fever, terminating in recovery after the temperature had gone to 110.8°. During the onset of the disease, a rash may be found, but this is an inconstant finding. It is of a macular type, appearing on the trunk and extremities, and usually lasts about one or two days and does not recur with subsequent relapses. The face is flushed, and the skin is hot and dry. There is a mild conjunctivitis, and the tongue is coated. The liver and spleen may become enlarged during the relapses, and although this is common, it does not necessarily have to occur. Jaundice is uncommon. There may be an associated bronchial cough which disappears with the fever. Urinary retention may occur. The pulse is stated to be rapid in most of the cases, although in several cases reported, the pulse was slow in the initial stage of the disease. With the relapses it goes to between 100 and 130. The white count may be low, normal, or high. There may be a relative increase in lymphocytes during each relapse. The urine shows findings consistent with fever: albumin and casts. Reynolds[12] reports that the Wassermann reaction is positive in 20% to 50% of these cases, but becomes negative with the disappearance of the disease. After recovery from the infection there is usually considerable weakness. However, other sequelae have been reported[12], such as hemorrhagic nephriti, iritis, cranial nerve paralysis, meningitis, pneumonia, polyarthritis and parotitis. Abortion has occurred in pregnant women. Gillespie[6] reports hemorrhagic nephritis and iritis, and radiculitis of the third, fourth, and fifth lumbar roots, in one of his patients. These all cleared up on recovery from the disease.

DIAGNOSIS

This disease is suggestive of several other conditions that must be ruled out: typhoid, undulant fever, malaria, rat-bite fever, and several others. This can be differentiated only by blood smear. It is important to obtain the blood smear just as the temperature is beginning to rise, either early in the disease or in a relapse, for very frequently during the course of the febrile period nothing can be found in the blood stream, and the blood is practically always devoid of spirochetae during the remission. [1]Evenly smeared blood slides taken as the fever rises during a relapse should be submitted. "They may show more than fifty spirochetae per field or only one in many fields."

TREATMENT

Neoarsphenamin is specific, and it is important to give this drug during the febrile period, preferably just as the temperature is beginning to rise during one of the relapses. Quite often one dose of .3 gram of Neoarsphenamin is enough to check an attack if given at this time. However, Graham[7] reports one of his patients relapsing after a ten-day interval following intravenous Neoarsphenamin. He was given .6 gram, and fourteen days later relapsed again. More treatment was given; he was free from symptoms for twenty-one days,

and then relapsed, requiring more Neoarsphenamin. Varden's[13] case relapsed, requiring more Neoarsphenamin. Burns'[4] case also relapsed after the initial injection. Irvine's[8] case relapsed after the initial injection. Following the administration of Neoarsphenamin, the temperature gradually subsides in twelve to twenty-four hours and the patient may become somewhat nauseated. When a patient relapses, one feels that he should continue treatment for some time to guard against other relapses. However, this probably has little value, since the most effective means of reaching the spirochetae is when they are in the blood stream, and this is found only during febrile periods.

CASE REPORT

CASE: E. M. J., Age. 32. This man had always been well. He went to San Francisco to see the World's Fair from June 15th to July 1st, 1939. On July 15th and 16th he went up to the White Mountains, around the vicinity of Greer, fishing, then returned to Phoenix.

He felt very well until July 23, 1939. On that evening he was tired, and did not care about eating. He felt about the same way the next morning; however, he went to work. During the morning he felt cold. The generalized aching began to increase. By two P. M. he had to go home because of aching and fever. On the 25th, his temperature went higher; his aching became more severe, particularly in his head, back, legs, and arms, and he vomited. He felt chilly but had no chill. He had no cough, no cold; he had no sore throat; his bowels were all right; there were no urinary symptoms. He complained only of the aching.

Physical examination at this time revealed: skin to be rather flushed; sclerae were moderately inflamed; nose normal; tongue was coated; throat moderately inflamed. Neck showed no adenopathy and no stiffness. Chest was clear throughout. Heart sounded normal, but the rate was only eighty. Abdomen, liver, and spleen were not palpable, and there was no tenderness. Extremities were perfectly normal; Kernig was negative. Temperature 103.8°. Respirations were easy; not increased.

Blood count: R.B.C. 4,700,000; hemoglobin 95%; W.B.C. 4,000; polynuclears 63%; lymphocytes 33%; monocytes 1%.

Impression: I thought this acted very much like influenza, with a low white count, slow pulse, and high temperature.

He was treated symptomatically. The next day there were still no physical findings. On the 27th, his temperature came down; he felt good. After remaining in bed about three days, he got up, went to work, and felt well until about ten o'clock in the morning, on the second of August, 1939. His temperature started up; he felt tired; he had some aching, but not as severe as before.

Physical examination was negative again except for a pulse of eighty, and temperature of 101.4°. Blood was taken for Widal and undulant fever tests; these were both negative.

On the third of August, his temperature was 103.6°, his pulse 96. Still nothing could be found. A smear was obtained for malaria, and none was found. His temperature dropped on the night of August the third. He was kept in bed, but on August sixth, his temperature went up again. It stayed around 101° during the day, and about three A. M., August seventh, it went up to 105.4° and then rapidly dropped by crisis. It left him feeling well, except that he was weak. This time his white count had gone up to 10,500 with polynuclears 60%, lymphocytes 27%, and monocytes 13%.

Urine: 1.023; albumin and sugar negative; no casts or blood cells; occasional white blood cell.

This was the third episode of fever. Relapsing fever was considered, and a smear taken on the seventh was examined by the laboratory for spirochetae, but none were found. Patient was kept in bed, feeling very good until about four P. M. August 15, 1939, when his temperature started up. A smear was immediately obtained, submitted to the laboratory for examination, and the spirochetae of relapsing fever were found.

He was given immediately .3 gram of Neoarsphenamin intravenously. That night his temperature went on up to 103°, but came down to normal the next night, when he was again given .3 gram of Neoarsphenamin.

He was given .3 gram of Neoarsphenamin intravenously again on August 20, 1939. Patient felt very well, and was working right along and was considered cured. However, on September 15, 1939, just one month after his first intravenous Neoarsphenamin, he began feeling tired. That evening, he began to ache, felt chilly and his temperature was 101°.

Physical examination was negative again except for the flushed skin, and injection of the sclerae. Some blood was obtained for smear, and the laboratory again found the spirochete of relapsing fever. He was given .3 gram of Neoarsphenamin immediately, and in order to prevent another recurrence, he was given five injections of .3 gram of Neoarsphenamin.

Since bismuth is effective against the spirochete of syphilis and the spirochete of Vincent's, he was given two injections of bismuth in oil, with the idea that this would last over a longer period of time. Up to the present, January 24, 1940, the patient has had no relapse.

CONCLUSION

Relapsing fever is a disease carried by a tick which transmits the spirochete from infected rodents to man. It has been found endemic in Texas, California, Colorado, and Nevada. Three cases have been previously reported from Arizona. Including the case presented here, four patients have been infected in the White Mountains near Greer, Arizona. Many people vacation in this region during the summers, and it is possible that other cases of relapsig fever have gone unrecognized, only because it has not been considered.

15 E. Monroe

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Symptomatology and Etiology of Spontaneous Hypoglycemia

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IN view of the widespread interest in spontaneous hypoglycemia since[1] the original investigation of insulin beginning in 1922[2], the demonstration of true hyperinsulinism due to excess function of islet cell tumors of the pancreas first proved in 1927[3], the recent detailed studies by the neuropsychiatrists of the manifestations of induced hypoglycemia in the shock treatment of mental disease, and[4] the rather enormous literature on the subject that has accumulated during the past decade, the title selected for this paper may well seem to be repetitious and even superfluous. As a clinical entity, because of its mimicry of so many other better known conditions, spontaneous hypoglycemia is still, no doubt, often overlooked. It is one of the conditions that is demonstrated only when kept in mind and appropriate investigations are undertaken. Although all the minute details of the regulation of carbohydrate metabolism are still not understood, the enthusiastic researches of the past few years have increased our knowledge considerably and we are in a much better position to evaluate this specific problem than we were able to do but a short decade ago, perhaps a further justification for reviewing the subject at this time.

SYMPTOMS

Since, of all the tissues in the body, the central nervous system is apparently dependent upon carbohydrate predominantly for its metabolic needs, it is not surprising that symptomatically the hypoglycemias express themselves essentially as neuropsychiatric problems. In the initial stages and less severe forms disturbed function of the autonomic nervous system is usually dominant. This is consistent with the belief that the first defense against a failing glucose supply is a heightened activity of the sympathico-adrenal system. Vasomotor disturbances such as pallor, palpitation, a sensation of painful constriction in the chest, sweating, pilomotor activity and a sense of tremulousness suggest the response to an overdose of adrenalin. At other times gastrointestinal symptoms such as hunger, epigastric discomfort or pain, or even vomiting, may be more disturbing and suggestive of the presence of a peptic ulcer.

Again, fatigue, nervousness, sweating, tachycardia and tremor may closely simulate the typical symptoms of hyperthyroidism.

As the blood sugar drops rapidly or reaches unusually low levels there is evidence that the oxygen consumption of the nervous system diminishes very appreciably, indicative of a greatly decreased metabolic activity approaching and simulating very closely the results of anoxemia. Under these conditions the patient may exhibit any conceivable variety and combination of neurologic and psychiatric disturbances. To attempt to enumerate all the possible variants in psychic or motor misbehavior would be to undertake an endless task. Among the milder manifestations are those seen in certain individuals who become suddenly and unjustifiably tired, exhausted and faint. This acute fatigue state, usually occurring some hours after the taking of food, may be accompanied by varying combinations of tremor, perspiration, palpitation, visual disturbance, muscular incoordination, mental slowness, impaired power of concentration, anxiety, confusion, depression and drowsiness. Again the mood may be quite different with a "sense of lightness", euphoria, volubility, unprovoked laughter, and so forth, suggestive of a happy stage of alcoholic intoxication. At other times the victim may be unreasonably irritable, quarrelsome, combative and negativistic. Paresthetic tingling of the lips, tongue, fingers, or toes are not uncommon early symptoms. Processes of thought and speech may be greatly slowed, or the subject may go about as though dazed and unaware of his surroundings and actions—typical somnambulism. At times these patients may commit acts of destruction or violence even to their dearest friends or relatives. In short, during an attack, they may behave like neurasthenics, hysterics, alcoholics, or like victims of any one of the major psychoses.

Of neurologic manifastations one may observe diplopia, nystagmus, variation in the size and reaction of the pupils, transient weakness or paresis of groups of muscles such as facial weakness or even complete hemiplegia. As the attack progresses there may be muscular twitching, rigidity even to opisthotonos, diminished consciousness from stupor to deep coma, and convulsive seizures, generalized or Jacksonian in type. Headache may be a prominent symptom either early or following an attack. Vomiting is not uncommon. During the more profound seizures there may be marked alteration in the tendon reflexes, and positive Babinski and Oppenheim signs are frequently observed. Sweating may be extreme. The temperature may fall to remarkably low levels while, on the other hand, after an unusually severe and prolonged attack, there may be a hyperpyrexia. If the patient escapes the diagnosis of some functional nervous disorder or a psychosis, he may well be suspected of having epilepsy, encephalitis, a brain tumor, a cerebral vascular lesion or some other form of organic nervous system disease.

SEQUELLAE OF ATTACKS

The attacks may terminate spontaneously, but are typically relieved by suitable carbohydrate taken either by mouth or intravenously. A more or less total amnesia for the more severe episodes is

Read before the New Mexico Medical Society, Gallup, N. M., May 11-13, 1939.

the rule. Unless the attacks are too severe and too closely repeated the patient appears to be quite normal during the intervals between. Following major seizures headache and drowsiness may persist for some time. Persons subject to repeated severe attacks may develop personality changes, becoming depressed, irritable and psychasthenic. There may be progressive mental deterioration. Such manifestations as paralysis, hemianopsia and aphasia usually disappear gradually, but may be prolonged. If the attacks are too severe, too prolonged, or too rapidly recurring, death may ensue and is apparently due to the production of irreversible degenerative changes which have been found to involve varying areas of the cerebral cortex and the basal ganglia. When this process has gone too far no amount of glucose will rescue the unfortunate victim.

The behavior of a patient in the midst of an hypoglycemic attack furnishes the most dramatic evidence of the vital importance to the organism of a steady glucose supply. The demands of the tissues for glucose depend upon their activity or metabolic rate of the moment. Obviously this demand varies greatly from hour to hour. The intake of food is ordinarily intermittent and also variable. Mealtime does not necessarily coincide with the period of greatest need. In spite of these things, under normal conditions the blood sugar varies within relatively narrow limits.

REGULATION OF SUGAR LEVEL

The regulators of this remarkable "homeostasis" may be grouped into three main divisions, though their functions are so interwoven that an actual separation is hardly possible. These are the endocrine glands, the autonomic nervous system, and the liver.

Of the endocrines, insulin apparently acts primarily in the conversion of glucose into glycogen in the peripheral tissues, thereby making it available for the production of energy. It also inhibits the too rapid release of glucose from the liver and may be necessary for the synthesis of liver glycogen. Its action, therefore, is to remove glucose from the blood stream and if it is ineffectually opposed the sugar content of the blood tends to drop toward zero. The other endocrines, and especially the adrenals, the thyroid and the pituitary have an opposite effect. The first defense against depletion of the blood's sugar is apparently adrenalin which increases the rate of glucose release from the liver and may actually inhibit temporarily the use of glucose by the peripheral tissues. The early symptoms of the hypoglycemic reaction appear to be due in large part to a sudden adrenalin release. The internal secretion of the adrenal cortex by a different mechanism also opposes the effects of insulin, but seems to be less abrupt and more continuous than adrenalin. An even powerful opponent to insulin is the pituitary. Without the pituitary present animals develop only a very mild diabetes following pancreatectomy, and persistent injections of a suitable pituitary extract have been shown to produce a typical diabetes with hyperglycemia, glycosuria and ketosis even in the presence of an intact and normal pancreas. Either by their effects on the function of the previously mentioned glands, the gonads and possibly the parathyroids may also influence carbohydrate metabolism.

Correlating the functions of the various endocrines is the autonomic nervous system. From the sugar which is located among the basal ganglia and closely interrelated with the pituitary by innervation and circulation, fibers extend by way of the vagus to the islands of Langerhans, and stimulate them while others over the sympathetic system activate the adrenals and the thyroid. This center is, no doubt, activated itself in one direction or the other by the sugar content of the circulating blood.

The liver is the central organ responsible for carbohydrate regulation. Here absorbed carbohydrate is stored and lactic acid, certain aminoacids and even products of fat metabolism are also converted into glycogen. This stored glycogen is later released as blood glucose, depending upon the needs of the moment. These functions of the liver, glycogenesis, glyconeogenesis, and glycolysis are controlled and regulated by the balanced activities of the endocrines and the autonomic nervous system. Under fasting conditions the liver seems to be the only source of blood glucose.

With this very brief and superficial resume of normal physiology in mind it becomes obvious that variations in the blood sugar content, in either direction, may occur when there are significant alterations in structure or function at any point in the controlling mechanism. In searching for the etiology of hypoglycemic symptoms, therefore, one must bear in mind the numerous possibilities for such a disturbance. For convenience of classification, the following systematic arrangement may be useful.

CLASSIFICATION

I. *"Physiologic" hypoglycemia.* It has been repeatedly demonstrated that the rate of utilization or storage of sugar can be stimulated and accelerated by the persistent intake of a relatively high carbohydrate diet. If the blood sugar curve of such an otherwise normal individual is followed for three or more hours after a carbohydrate meal, such as 100 grams of glucose, it is not infrequently found that, following a moderate rise during the first hour, the level may fall to quite a low point—to 70 mg. or less. There may be no reaction noticeable or, at the time of the most rapid drop the subject may complain of hunger, and may experience some faintness, nervousness, palpitation or sweating, and there may be headache. Such symptoms will usually disappear gradually as the blood sugar again rises. Food will give prompt relief. A large percentage of people have, no doubt, experienced such mild symptoms on occasion. They may

be induced also at times by unusually prolonged exercise or by too long a delay in the time of the accustomed meal. This type of hypoglycemia does not tend to become progressive and usually has no serious import. Such observations should remind us, however, that the finding of a moderately low blood sugar alone does not necessarily mean that we have arrived at a diagnosis of a truly pathological condition. The recognition of the stimulating effect of carbohydrate has been of service therapeutically. A goodly number of patients with annoyingly repeated and even severe hypoglycemic reactions have been improved by encouraging them to follow a relatively low carbohydrate and correspondingly higher fat and protein diet.

II. *Hypoglycem'a due to lack of opposing secretions.* As previously indicated, the pituitary, the adrenals and the thyroid have, through their hormones, an effect on the blood sugar opposite to that of insulin. Hypersecretion of these organs tends to produce hyperglycemia, while severe degrees of failure have an opposite effect. Destructive lesions of the adrenal cortex, as in Addison's disease, are frequently accompanied by chronic hypoglycemia, and the patient may actually die in hypoglycemic coma. Similar conditions may at times be observed in subjects of pituitary cachexia, Simmond's disease, or following destructive pituitary tumors. Likewise, in association with severe degrees of hypothyroidism the blood sugar may be chronically or periodically low, though severe symptoms attributable to hypoglycemia are apparently less frequent than in association with adrenal or pituitary failure. In this connection it should be remembered that in the presence of hyperthyroidism there may be occasional hypoglycemic periods in contrast to the more usual hyperglycemia if, as a result of continuous overstimulation, the store of liver glycogen should become exhausted. Such a condition has been observed following operations on toxic thyroids with serious consequences.

In this group the low blood sugars often found may be attributed to a relative insulin excess but there is no evidence of any disturbance of structure of the islands of Langerhans. All these patients are highly sensitive to injections of commercial insulin.

III. *Hypoglycemia due to dysfunction of the nervous system.* Since the observations of Claude Bernard it has been recognized that destructive lesions involving structures located in the floor of the third ventricle may cause hyperglycemia and consequently glycosuria. Only within the past few years has the opposite effect been recognized. Vagal overstimulation, as opposed to sympathetic hyperactivity, would appear to be the logical mechanism. A great difficulty in distinguishing cause from effect arises in consideration of this group since, as previously indicated, the symptoms and signs of significant hypoglycemia from whatever cause are chiefly manifestations of disturbed nervous system function. Early there is evidence of sympathetic over-activity; later, in profound attacks, there may be complete disorganization of autonomic balance with vagal symptoms predominating. Added to these are variable symptoms indicative of cortical damage and evidence of definite involvement of any or all the basal ganglia. Temporary or permanent signs of focal damage, such as aphasia or motor weakness, only serve to confuse the issue. Yet occasional observations of hypoglycemic reactions in association with brain tumors in certain locations, occurring with or producing the "seizures" of general paralysis, following encephalitis, and so forth, would appear to be adequate evidence that irritative lesions in the region of the vagal centers may force the blood sugar down possibly by an overstimulation of insulin production. It has been observed repeatedly that persons with an unstable autonomic nervous system, especially the vagotonic, hypotensive type, are particularly subject to periodic symptoms such as we have attempted to describe, and that these symptoms are associated with a low blood sugar. Furthermore, these individuals may have very low blood sugar curves following the ingestion of glucose. Low carbohypdrate diets with shortened intervals between meals and general measures directed to improve their general physical and nervous state have been quite successful therapeutically. It is somewhat an open question as to whether the nervous imbalance predisposes these individuals to periodic or chronic hypoglycemia, or hypoglycemia from some other cause is provocative of their nervous reactions. This group offers a most interesting field for further investigation.

Whether to classify the hypoglycemic symptoms not infrequently observed in some women in association with the menstrual periods in this group or in the previous group of primary endocrine disturbance is difficult to answer at this time. The close integration of endocrine function and the autonomic system may well render an attempted division of this type somewhat superfluous.

IV. *Hepatogenous hypoglycemia.* Animals deprived of their livers die from hypoglycemia unless measures are taken to provide a continuous glucose supply. Clinically, any disease process that seriously interferes with the functions of glycogenesis or glycolysis may deprive the blood stream at times of adequate sugar. This has been observed in various forms of acute hepatitis, infectious or toxic, in extensive carcinomatosis, in extreme fatty infiltration such as may be associated with alcoholism, in advanced cirrhosis and in chronic infectious hepatitis. Von Gierke's or glycogen storage disease is characterized by hepatomegally and hypoglycemia. In this condition the liver is filled with glycogen, but the process of glycolysis is seriously impaired.

It appears that in the past too little attention has been given to disordered states of liver func-

tion as significant factors in the production of clinically important hypoglycemic manifestations. These same patients may have alternating periods of hyperglycemia and glycosuria simulating and often confused with diabetes.

Other conditions tending to deplete the glycogen stores of the liver may be listed in this category, such as the hypoglycemia of starvation and of prolonged strenuous exercise, as in the classic example of the marathon runners. Physical exertion tends to exaggerate any tendency toward hypoglycemic reactions, whatever the etiologic background.

V. *Hypoglycemia due to island cell tumors of the pancreas (true hyperinsulinism).* Of all the spontaneous hypoglycemias, the most dramatic and the one which has received the most enthusiastic attention is that resulting from an excess insulin production of tumors, malignant or benign, arising from the island cells of the pancreas. Recognized first but 12 years ago, these adenomata have now been reported from most parts of the world. While the early symptoms may be mild and relatively infrequent, they tend to become progressively severe and disabling. On the other hand, the first attacks may be suddenly violent. In the presence of these tumors all methods of conservative management eventually fail. With successful surgical removal a complete cure can be expected. It must be remembered, however, that more than one adenoma may be present. Surgical cure has now been accomplished in a considerable number of instances. Fortunately only a relatively small number of proved tumors have been definitely malignant with evidence of metastases. The possibility of malignancy, however, is one important reason why surgery should not be unduly delayed if one can convince himself that the hypoglycemia is not due to some extra-pancreatic disturbance.

These adenomata are often quite small and difficult to find even after complete mobilization of the pancreas. In two patients whom I have had the opportunity to observe, the small tumors were located in the posterior portion of the pancreas so far toward the duodenal end that they were missed at operation even after careful search. One patient was explored twice, but the tumor was discovered only at autopsy. In third patient the small tumor was easily visible on the anterior surface and was removed with entire relief from symptoms.

In the newborn and young children, especially those of diabetic mothers, a generalized hyperplasia of the islands of Langerhans with hyperinsulinism is occasionally seen. This condition has been but rarely demonstrated in adults.

VI. *Hypoglycemia of unknown etiology.* For the sake of completeness it must be admitted that in some instances, even of severe degrees of hypoglycemia, no satisfactory explanation of the condition has been demonstrated even after meticulous study, surgical exploration, and even subtotal pancreatectomy. Time will no doubt supply the answer.

DIAGNOSTIC CRITERIA

A brief word only about diagnosis: Before we decide upon a diagnosis of spontaneous hypoglycemia or hyperinsulinism, it should be definitely demonstrated that the patient's symptoms coincide with the presence of a low blood sugar, typically 70 mg. or considerably lower, that he can be relieved by glucose or other suitable carbohydrate and that typical symptoms can be precipitated by starvation or by the administration of a small dose of commercial insulin. Numerous observation have been made to indicate that a rapid drop in the blood sugar is more apt to produce a typical reaction than a gradual fall even to low levels. For this reason there is no definite correlation between the actual blood sugar content and either the onset or severity of symptoms. In hypoglycemic coma, however, the blood sugar is likely to be below 45 mg. per 100 cc.

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Annual Meeting

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NEW MEXICO MEDICAL SOCIETY

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Albuquerque

May 27-28-29

SOUTHWESTERN MEDICINE

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MASS MEDICINE?

Laudable is the present day interest of the public in preventive medicine. For years the medical profession has fostered the development of such on interest. Now, with public cooperation, the various health services are showing remarkable statistical improvement in the field of disease preventiond. The majority are being well cared for. Further radical improvement may be dependent on departure from today's mass methods.

Educational campaigns, posters, lectures and miscellaneous publicity efforts bring many people into the public health clinics. There they are exposed to advice, admonitions, persuasion. There also they receive mass immunizations. Yet many of these, plus other persons, not clients of the public health service, later become numbers on the wrong side of the health ledger. In other words, too many people still get sick.

Now, here is a field of work for the family doctor, despite the sneers of the self-anointed Hugh Cabot. Nearly any physician, aside from the institutionalized automaton, well knows that, due to something inherent in the individual, certain people seem bound to contract certain diseases, especially herded in masses as targets for usual health measures. And there is the point of this discussion —the individualism of human beings in their response to environment. We note that certain cases, say of tuberculosis, get well easier and faster than other cases whose objective findings may be identical with those of the first group. Then we say "strong constitution." We have seen certain children in a family contracting poliomylitis, while their brothers and sisters remain well. Then we say "immunity." Everyone is aware that nervous make-up and mentality are greatly concerned in any case of organic pathology, and the response of the entire organism to that pathology. Prescribers beware of idiosyncrasies to various drugs.

The prevention and treatment of disease in the individual must forever remain a peculiarly individual, highly personalized problem. The solution does not lend itself to mass methods, if a thorough, complete, honest solution is to be obtained. Mass methods are like a pint of strawberries for a regiment—fine as far as they go. Then must enter personal, individual attention by the family doctor to the individual being. He must see his patients in four categories: (1) morphological (2) physiological (3) immunological (4) psychological. He must know how his patient's body is shaped, how it works, how it resists infections, how it thinks and feels. Knowing these things, he is in position to advise, to prevent or treat disease. And who can be in better position.

Human beings cannot yet be treated as numbers of interesting cases of this or that. They must be seen and cared for as highly organized, complex, individuals. Medicine still needs the family doctor. More, the public needs him. No one else can do the job—least of all the belligerent, prejudiced advocate of mass medicine for the masses.

WAR AND MEDICINE

Wilder Penfield, addressing the McGill Medical Undergraduate Society, has this to say[1]:

"The sound of marching feet across the Polish frontier changed the world in which we live. For every man war is a different thing. It is one thing for us in Canada and another for the man in Polandl, in England, in Germany. It is one thing for a ma in civil life and another for a man in medicine. What does this war mean to a medical student?

I may as well admit at once that I was a medical student when the last was broke out and that gives me a measure of understanding of your dilemma at this time. You have probably had the impulse to throw down books and test tubes and march in a combatant company, but you must first stop to consider the need for medical men in this crisis. We who have our training are ready and will go into the Army Medical Corps when needed. But you, too, will be needed as doctors before this affair is over.

Perhaps in the mirror of history you can see yourselves in a truer perspective against the background of this war. 'History', Macphail said, 'is the master to whom we all must go.' Advances in medical science have done much to change the face of warfare. Until recently contagious disease was wont to follow each army about, destroying more men from behind than the enemy could kill from in

front. Now, however, typhoid fever, smallpox, typhus, malaria, and to a large extent even syphilis have surrendered to inoculation, vaccination, delousing, sanitation and prophylaxis. The problem of more rapid and efficient handling of the wounded remains to be solved with a thousand other lesser problems to which only the fully trained medical man can contribute.

If this war continues now, which God forbid, I have tried to point out that Canada can ill afford to allow her medical schools to decrease the normal supply of doctors nor to lower their standards of instruction. You have received cards on which to signify to Ottawa your willingness to serve wherever you may be needed. I have no doube your signatures were in the affirmative. That being the case, you will be notified if new conditions alter the national needs. Your task, in the midst of turmoil, is to study medicine quietly and with the singleness of purpose that breeds greater accomplishment. Short cuts may be found to your medical degree, I do not know, but they must not result in less thorough training. Look at the war news if you must, but abjure the bewildering blare of radio broadcasts and resolutely ignore what you cannot forget.

In this war only a coordinated effort that takes advantage of the abilities of specially trained men can succeed. In this effort there is no place for personal pride or preference. Let the anatomy and physiology of man be your concern. It is so simple when compared to the explanation of this folly. Your immediate task is to carry on."

Thus does war come home to our neighbor on the North, land of such medical great men as Osler, Banting, Best, McCleod, Penfield and Collip. Thus bravely and in the best Anglo-Saxon tradition of devotion to duty and the ideals of free man do the honored men of Canada meet the latest challenge to their way of life. May American medicine never have to face the dark nights in store for our brothers in Canada. But if the dread gauntlet be flung, may we as unanimously respond in our adaptation as our sorely beset neighbors of the friendly Dominion.

1 McGill Med. Jour., Feb. 1940.

ALBUQUERQUE SESSION

Elsewhere in this issue there will be found the program for the New Mexico Medical Society's annual session at Albuquerque May 27, 28 and 29. Headquarters will be at the Hilton Hotel. The registration fee of $5.00 includes admission to all sessions, the two luncheons, the dinner-dance and the smoker.

Dr. George T. Colvard, of Deming, will retire as president and be replaced by Dr. W. B. Cantrell, of Gallup. General chairman for the meeting is Dr. B. F. Roberts, of Albuquerque. The scientific committee is composed of the following: Drs. M. K. Wylder, chairman, J. D. Lamon, J. W. Hannett and R. W. Mendelson. The social and entertainment committee is headed by Dr. H. L. Brehmer. Chairman of the financial committee is Dr. Carl Mulky. Dr. M. P. Beam is chairman of the committee of reception and registration.

The House of Delegates opening session will be on Monday morning, May 27, at 9:00 a. m. One hour later the scientific program begins. The meeting is scheduled to end at noon, May 29. Mayor Clyde Tingley, of Albuquerque, is to deliver an address of welcome. Scientific papers are to be given by:

Dr. Carl Maynard, Pueblo, Colo.
Dr. Willis C. Campbell, Memphis, Tenn.
Dr. Frederick Witherell, Syracuse, N. Y.
Dr. J. L. Emmett, Rochester, Minn.
Dr. Ben Massey, Pasadena, Calif.
Dr. E. M. Lazard, Los Angeles, Calif.
Dr. Albert Soiland, Los Angeles, Calif.
Dr. J. R. Jaeger, Denver, Colo.
Dr. Paul Bassell, Temple, Texas.
Dr. Rupert Raney, Los Angeles, Calif.
Dr. R. M. Balyeat, Oklahoma City, Okla.
Dr. Dan Stine, Columbia, Mo.
Dr. Richard Sutton, Jr., Kansas City, Mo.
Dr. Berne Hunt, Los Angeles, Calif.
Dr. A. L. Levine, New Orleans, La.
Dr. C. N. Giere, El Paso, Texas.
Dr. George J. Pattee, Denver, Colo.
Dr. Paul J. Conner, Denver, Colo.
Dr. W. W. Barber, Denver, Colo.
Dr. O. J. Farness, Tucson, Ariz.

Most of the papers are to be discussed by physicians from New Mexico and El Paso. There will be 12 commercial exhibitors and a number of scientific exhibitors. The New Mexico State Public Health Laboratory, the U. S. Indian Service and Carrie-Tingley Hospital are to have rather elaborate exhibits.

There will be entertainment for the visiting ladies and also plenty of entertainment outside of instruction for the physicians in attendance. Thi is the first time that Albuquerque has entertained the New Mexico Medical Society in several years. With the amount of new construction in Albuquerque there has been an increase in facilities for caring for large conventions. This year's scientific program is exceptional in its scope and the calibre of speakers selected to serve as instructors. The session promises to be one of the most valuable in the history of the New Mexico Medical Society.

SOUTHWESTERN MEDICAL ASSOCIATION

At a recent meeting of the Executive Committee of the Southwestern Medical Association, under the direction of Dr. Orville Egbert, president, dates for the fall meeting in Tucson were set to be November 21, 22 and 23. The official hotel will be the Pioneer Hotel.

Dr. Egbert has appointed the following Program Committee: Chairman, Dr. C. A. Thomas, Tucson; Dr. Dake Biddle, Tucson; Dr. C. S. Kibler, Phoenix; Dr. Leslie Smith, El Paso; Dr. E. W. Fisk, Santa Fe; Dr. J. M. Greer, Phoenix. Dr. C. A. Thomas is also chairman of the session. The Program Committee has been allotted a larger sum of money for its budget this year than has ever been so allotted before. The work of building this fall's scientific program is well under way.

Special Section

Arizona State Medical Association

PRESTON T. BROWN, M.D., *Associate Editor*
403 Professional Bldg., Phoenix, Arizona

*AGRICULTURAL WORKER HEALTH & MEDICAL ASSOCIATION

H. D. KETCHERSIDE
Phoenix, Arizona

I have been asked to report the work of the Agricultural Workers Health and Medical Association.

I was surprised to find that many of our members did not know of the existence of such an organization, how it came into being, and what class of people it serves. Therefore I feel called upon to start at the beginning and outline the problem as well as to report on the efforts being made to meet it.

The transient agricultural worker has become a major problem only in recent years; before that time the transient laborers, in Arizona and California, consisted mostly of Mexican families, and single men. The Mexican families usually returned to Mexico when the cotton picking was completed, and the single men, being unencumbered, caught a freight train and moved on. They were not then a serious problem and are not today. Neither were the class of people who are now our chief problem considered as a social problem by the states in which they resided. For generations they had been tied to the soil and were a necessary part of the social economy of their states. They were mostly sharecroppers who managed to eke out a bare existence on 40 acres of cotton land and return a nice profit to the landowners. Very few of them were ever recipients of any form of relief. They were of good old American pioneer stock, the descendants of Daniel Boone, Davy Crockett and Sam Houston, and many of them are eligible for membership in the Sons and Daughters of the American Revolution. The difference between them and most of us here present is mostly a difference of opportunity. It is true that the hopelessness of their plight and failure to rise above it has killed much of their ambition, but this can and must be restored before any real solution of this problem can be hoped for.

What happened which changed this picture so suddenly? First, the price of cotton dropped until the sharecropper could not make a living on 40 acres and return any profit to the landowners. It became necessary to dispossess the sharecroppers and farm the land in large acreages with tractors. These people were forced to pack their belongings and their families in old cars and start looking for work, which not to be had, for the same process was going on over a very large area.

How did they happen to come to Arizona and California? We invited them. The Arizona Cotton Growers, through newspaper articles in their home states, handbills and posters, invited them to come to Arizona, promising them higher wages and desirable working and living conditions. Of course, they came in overwhelming numbers. How they were received I believe most of you know. They could not be driven out because they had spent their last cent to get here and they no longer had any place to go. Since that time they have been moving back and forth between California and Arizona.

Of course, these people had medical problems many more than our fixed population. How did we meet these problems? Mostly by ignoring them. No one felt that the problem was theirs. It is true that many of the doctors gave unstintingly of their time without promise or hope of remuneration, but it was almost impossible to get them into a hospital and as these people had no homes the treatment was very unsatisfactory.

It was felt that these people were not our people, and the federal government, through our senators, was asked for help. The problem was accepted, and the Agricultural Workers Health and Medical Association was established in California in the spring of 1938, and extended to Arizona in November of that year, with funds obtained from the Farm Security Administration.

You should know that this organization did not immediately plunge into medical rellief. The men picked to head the organization realized, as I believe most of our representatives in Washington have come to realize, that no medical program can hope to succeed without the full co-operation of the medical profession. They came first to the medical societies of the two states, presented their problem and asked for advice, and they have at all times followed the advice given.

Since the migratory farm labor population in Arizona is concentrated in Maricopa County, 84% of the cotton acreage of the state being in that area, the state medical association allowed the Maricopa County Medical Society to work out the plan, and a committee was named, of which I happened to be chairman. This committee met many times, and a fee schedule was worked out between the two states, which was understood by everyone concerned to be a substandard schedule based on the fact that this was an emergency relief program. Rules were made to see that the patient had

Read to the House of Delegates, Arizona State Medical Association, April 17, 1940.

The President's Page

IN this, my final official communication, I desire to voice my deep feeling of gratitude for the splendid cooperation given me during my tenure of office. Without this spirit of cooperation our accomplishments during the year would have been practically nil.

I am also grateful for the privilege given me to serve as your President—an honor that can come to but relatively few of the many qualified members of this Association. No man could be selected for this office of great responsibility and opportunity for service without being fully aware of his own limitations and the realization that whatever success he attains depends almost entirely on the whole-hearted cooperation and support of those with whom he is associated.

With the aid and support of YOUR Council, I have earnestly and conscientiously endeavored to conduct the affairs of OUR Association in a sound and businesslike manner, and I feel that an inspection of our records will verify the accuracy of this statement.

As we stand at the threshold of another year, we, as physicians, should look upon it as a period of retrospection. Not for the mere purpose of condemning our mistakes, but with the hope that by reviewing our past record we may be able to outline for the year about to be ushered in, a bigger and more noble plan of action. Each New Year in our Association should be to us a milestone, marking on the one hand our accomplishments and failures, and on the other hand pointing as a guiding finger to the broad expanse of future endeavor that awaits us. The question of the hour should not be, what have we accomplished, but what are we going to accomplish. Our failures and mistakes should be but a spur to a more glorious record for the years to come.

May I urge that you continue to give the incoming officers of this Association the same helpful and friendly cooperation which you have most graciously extended me in order that YOUR Association may continue its march forward?

In all sincerity,

Chas. S. Smith M.D.

CHARLES S. SMITH, M. D.

free choice of physicians, and since most of these people did not already have a physician, rules were made to see that the work was distributed as evenly as possible. At the request of the organization, after the plan was put in operation, the committee from the county society was continued as a permanent advisory committee. As the program expanded into Pinal, Yuma and Graham counties, the plan as instituted in Maricopa county was adopted as a whole.

During the first year of operation in Arizona there was spent for medical relief $199,499, of which the physicians received $91,157, dentists $4,003, nurses $970, hospitals $60,924, druggists $16,719, medical supplies $15,615, clinic fees to physicians $3,001, clinic drugs $2,329, and miscellaneous expense $4,412.

During the period from July 1, 1939, to February 29, 1940, there has been spent for medical relief $355,226, divided as follows:

Physicians	$159,713.00
Dentists	8,932.00
Nurses	1,562.00
Hospitals	122,395.00
Drugs	22,136.00
Medical supplies	16,624.00
Special diets	537.00
Clinic fees	8,056.00
Clinic drugs	7,306.00
Miscellaneous expense	7,960.00

It seemed advisable for this organization to have a medical adviser in Arizona, and I resigned from the advisory committee to accept that position, but I wish you to know that we will continue to rely on the advisory committees in each county where we operate, to tell us how this organization should be run.

PROCEEDINGS OF EL PASO COUNTY
TUMOR CLINIC
March 12, 1940
EL PASO CITY-COUNTY HOSPITAL

Present: Drs. Cathcart, Causey, Gallagher, Hardy, Holt, Murphy, Newman, Peticolas, Waite and Powell; also Dr. Buhler of Kansas City.

Case 1. N. E., a 68-year-old Mexican man, was present at Tumor Clinic for the first time today. Case presented by Dr. Powell, as follows: This man first noticed a small postule in the left eyebrow and superior eyelid in May of 1938. In April of 1939 he came to the clinic and a biopsy was done. Dr. Waite reported no malignancy. Treatment before entrance consisted of bismuth and K. I. gtts. Examination on entrance showed an ulcerated tumor, very rough, with itching, 2x3 cm. in measurement, in the left brow and superior eyelid. There was fixation, ulceration and induration. No regional nodes. The lesion extends into the eyebrow and has everted the superior eyelid. Biopsy scar has pulled the eyelid up also. Discussion: Dr. Hardy questioned whether biopsy did any good, since the diagnosis depends so largely on clinical findings. He referred to a series of cases in another part of the country which were diagnosed malignancy by biopsy, but they lived too long and the diagnosis was changed to granuloma. Diagnosis: Ulceration of eyelid from infection following excision of the lesion. Recommendation: As soon as possible let that eyelid down by plastic surgery.

Case 2. T. T., a 40-year-old Mexican woman, with a tumor in the left supraclavicular region, was seen in Tumor Clinic today for the first time. Case presented by Dr. Powell, as follows: This woman first noticed a swelling in her neck in 1937. She came into the clinic in 1939, and was given SSKI gtts. The tumor was the size of a large walnut, in the left supraclavicular region. There was fixation. No ulceration, induration, roughness, bleeding or itching. No regional nodes. No similar condition in other parts of the skin. The mass is pulsating. It has never caused her pain. Discussion: Dr. Hardy remarked that the bruit was not very marked. Dr. Gallagher asked if the patient discovered the tumor herself and was told she had. Diagnosis: Aneurysm of left subclavian. Recommendation: No treatment recommended.

Case 3. V. B., an 18-year-old Mexican girl, with a small tumor of the anterior cervical region, was seen today for the first time in Tumor Clinic. Dr. Powell presented the case, as follows: This girl first noticed a small swelling in the neck September 1, 1939. She came to the clinic March 5, 1940. She had had no treatment before that. The tumor was submaxillary and pretracheal. No ulcer, bleeding, roughness, itching or pain. She had had no similar lesion previously. The tumor is about 2 cm. in diameter, indurated and attached to the skin. No regional nodes. Discussion: Dr. Gallagher—Sebaceous cyst; suggest cauterization. Dr. Holt—Branchial-cleft cyst. Dr. Murphy—(1) Most likely brachial cyst. (2) Ordinary sebaceous cyst. (3) Thyroglossal cyst. Recommend taking it out, being sure there is no connection with pharynx, hypopharynx, trachea or tongue. Dr. Cathcart—Branchial-cleft cyst. Recommendation: Removal (or possibly cauterization) and report at next tumor clinic.

Case 4. A. P., a Mexican man, age 75, with a tumor on the left side of the neck, was seen today in Tumor Clinic for the first time. Dr. Powell read the following report: This man first noticed a swelling on the left side of his neck February 12 of this year. He came into the clinic 10 days later. The tumor was about 5x2 cm. in size, and appeared to be cystic. Patient complained of slight dyspnoea and referred pharyngeal pain, and hoarseness. A biopsy was done and the laboratory reported papilloma. On questioning today, patient said the hoarseness came on suddenly 15 days ago. Discussion: Dr. Gallagher—This is entirely different from the last case. In don't see why a branchial-cleft cyst should be attached to the skin, for in-

stance. Dr. Cathcart—When they get ready to burst they inflame the skin. Dr. Gallagher—Yes, but that one is not getting ready to burst. Diagnosis: Dr. Cathcart—I think it is some sort of lymphatic stasis. Dr. Newman—I think it is a cyst. Dr. Peticolas—I think it is a cyst, but I think he has something else wrong with him, too, and I don't think surgery should be attempted at this time. Dr. Murphy—A mixed-cell tumor of the parotid. Dr. Hardy—Granuloma of some kind, possibly syphilitic. Dr. Holt—Some sort of cyst. Further discussion: Dr. Hardy—It might also be a Hodgkin's. Dr. Waite—He is too old for Hodgkin's. Dr. Hardy—The lower part of it contains fluid, but the upper part is entirely solid, I believe. It might be a gumma. He should have a Wassermann. Recommendation: Aspiration. To return to Tumor Clinic in a month.

The case of L. H., complete with autopsy report, was read by Dr. Powell. (Hospital Record No. 540.) Dr. Gallagher remarked that the working diagnosis (carcinoma of lung) was "a good guess, but not a diagnosis."

NEWS

General

The American Medical Golfing Association's twenty-sixth annual tournament will be held at Winged Foot Golf Club, Mamaroneck, N. Y., Monday, June 10, 1940. Some 250 out of the 1,350 Fellows of the A. M. G. A. are expected to take part at Winged Foot in the 36-hole competition.

Officers of the A. M. G. A. for 1940 are George Washington Hall, M. D., Chicago, president; D. H. Houston, M. D., Seattle, first vice-president; Grayson Carroll, M. D., St. Louis, second vice-president; Bill Burns, secretary, 2020 Olds Tower, Lansing, Mich.

All members of the A. M. A. are eligible for Fellowship in the A. M. G. A. For registration application write the secretary.

El Paso

The regular staff meeting of the Hotel Dieu Sister's Hospital was held Tuesday, March 5, 1940, at 12:10 p. m. in the auditorium of the Nurses' Home. Luncheon was served. The program was as follows: "Perforated Duodenal Uulcer; Subdiaphragmatic Abscess," by Dr. J. L. Murphy; discussion by Dr. James Gorman and Dr. W. W. Waite. "Bronchopneumonia; Coronary Thrombosis," by Dr. C. D. Awe; discussion by Dr. J. Mott Rawlings.

A regular meeting of the City-County Hospital Staff was held Wednesday, March 20, 1940, at 6:30 p. m., at City-County Hospital. The program was as follows: "Syphilis in El Paso," a panel discussion by Drs. Leslie Smith, Wickliffe Curtis, Robert Thompson and A. W. Multhauf; moderator, Dr. M. P. Spearman.

A regular meeting of the El Paso County Medical Society was held March 11, 1940, at 8:00 p. m. in the tea room of the Hotel Cortez. The program was as follows: "Low Back Pain and Sciatica," by Dr. E. W. Secord; "Recent Advances in Otolaryngology," by Dr. M. P. Spearman; "Maternal Mortality," by Dr. F. A. Snidow.

Dr. W. R. Curtis, El Paso, was recently notified that he is to be certified by the American Board of Urology, having passed its recent examinations.

AUXILIARY NEWS

The Women's Auxiliary to the El Paso County Medical Society was held March 12, 1940, at the home of Mrs. Frank P. Schuster. Mrs. Branch Craige, present, presided.

The following were elected to the board of directors for 1940-41: Mesdames N. H. Keller, A. D. Long, Travis Bennett, C. H. Mason.

The following are the newly elected officers: President, Mrs. James W. Laws; president-elect, Mrs. Henry Safford, Jr.; first vice-president, Mrs. A. P. Black; second vice-president, Mrs. Leslie Smith; third vice-president, Mrs. J. W. Cathcart; treasurer, Mrs. Jesson Stowe; recording secretary, Mrs. Jacob Rogde; corresponding secretary, Mrs. S. G. Von Almen.

Delegates were elected to attend the state meeting in Dallas in May, 1940 as follows: Mrs. Branch Craige and Mrs. Louis Breck; alternates; Mrs. James Laws and Mrs. George Turner.

Mrs. H. T. Safford, Jr., program chairman, then introduced Mrs. Irving McNeil, who reviewed the book, "Old Santa Fe Trail," by Stanley Vestal. Mrs. Samuel Rennick, in charge of the musical part of the program, presented Miss Mansfield in two piano solos. The meeting was adjourned and tea was served. The hostesses assisting Mrs. Shuster were: Mesdames H. T. Safford, Jr., M. P. Shuster, S. A. Shuster, Hugh Shannon, F. A. Snidow, M. P. Spearman, Erich Spier, Burleson Staten, B. F. Stevens, H. E. Stevenson, Walter Stevenson, J. L. Stowe, S. D. Swope, E. D. Strong, J. W. Tappan, S. L. Terrell, R. F. Thompson, George Turner, James Vance.

The next meeting, April 8, 1940, will be held at William Beaumont General Hospital. The hostesses will be the wives of the medical staff of William Beaumont General Hospital and Fort Bliss.

—*Malvina Spearman.*

MISCELLANY

ETHER CONVULSIONS

1. Convulsions under ether anesthesia are so unexpected and terrifying that we must have a definite plan of treatment agreed upon between anesthetist and surgeon.

2. Check temperature before anesthesia is started.

3. Adrenalin must be given instantly.

4. Soluble barbiturate must be on hand for instant use, whenever a child is being operated for an acute septic process.

5. Ether anesthesia must be stopped at once on appearance of premonitory symptoms.

6. Chloroform, oxygen and carbon-dioxide must be readily available.

7. Calcium gluconate or dextrose (50% intravenously) may be necessary to stop convulsions.—*Minn. Med.*

OCULAR HEADACHE

Before entering the first grade, every child's eyes should be refracted under atropine so that he can be protected against abusing a pair of inadequate or deficient eyes. The girl with the nervous breakdown, the child who is inattentive, the person in business who has a headache at noon, is relieved by lunch, and then has a recurrence about 3 or 4 in the afternoon, the clock-watcher, the student who cannot concentrate, the convalescent patient who has headache and is reading in bed—these and many others belong to the group of people who have faulty, poor, or inadequate eye musculature. They may require exercises, surgery, or glasses, or all three.

I can also tell you that many of these patients require medical treatment for a deficiency in thyroid or vitamin A. Some have general muscle exhaustion due to menopausal disturbance or some cerebral disturbance. It is well to remember that a patient with ocular muscle imbalance may have his primary disturbance elsewhere, the muscle trouble being secondary and the pain the tertiary portion of the entire picture. It is well to think of these patients as sick people who need more than a pill; they need careful study and definite management. Anything short of this fails to relieve the pain.—*Jo. M. S. M. S.*

BUT THE GREATEST OF THESE

The physicians of the United States contribute millions to charity. They render service to the unfortunate without thought of recompense. In their relation to the layman they are, in all probability, more charitable than any other professional group. The physician's charity to the underprivileged classes is not merely that of generosity from a monetary standpoint, or rendering service without thought of fee. Regardless of the race, color, creed or financial status of the individual patient, it is to the family doctor that he turns for understanding, for kindness, for charity, for confession.

If the layman prompts a charitable response from the physician, why, in general, is the same physician so uncharitable to his fellow colleagues? Why is he so critical and so free to give voice to criticism? Why does he impugn the motives of his fellow practitioner when he is so understanding of the mistakes of the layman? Why is he frequently unkind and bitter to his professional brother, yet philosophical when provoked by a layman?

Probably the greatest source of unpleasantness between physicians is possession of patients. For some reason the physician has acquired the feeling that his patient is his chattel, and if his patient consults another physician his chattel is stolen, and the battle is on. In squatter sovereignty "possession is nine points of the law," but in medicine possession can be retained only by virtue of the continued confidence of the patient in conscientious, skillful and understanding care. The question of medical ethics is frequently embarrassing to the patient, but regardless of ethics the patient is entitled to consult whatever physician he wishes, and his choice is based on confidence in the integrity and the ability of the individual doctor. In the oath of Hippocrates the physician swears by Apollo and Aesculapius to impart a knowledge of the art to disciples. The treatment accorded the young doctor who enters a community does not ordinarily comply with the Hippocratic oath, and charity and kindness do not characterize his reception.

The physicians of these United States have much to be grateful for. There is an abundance of work for everyone, and no one man can do all the work in his particular community. Harmony and understanding would prevail if the doctor would emulate the example of Paul, who taught, "Charity is patient, is kind; charity envieth not, dealeth not perversely; is not puffed up, is not ambitious, seeketh not her own, is not provoked to anger, thinketh no evil; rejoiceth not in iniquity, but rejoiceth with the truth."—*Jo Iowa St. Med. Soc.*

MINIATURE FILMS

The General Electric X-ray Corporation announces the successful "development of a reasonably accurate yet relatively inexpensive method of applying x-ray examination to large groups of people." The method consists of photographing the fluorescent image on 4x5-inch films. An extremely fast screen has been developed as well as a specially prepared lens capable of producing a very sharp image. The method is apparently superior to the use of sensitized paper, fluoroscopy and the smaller 35-mm. film.

At the last meeting of the Radiological Society of America, Potter, Douglas and Birkelo reported, in a comparative study of 1,610 chests, using the standard 14x17-inch and the new 4x5-inch films, results which indicate the surprising accuracy of

the new method. There was an error of only 2.6% in a total of 271 active lesions demonstrated by both methods. The new photoroentgenographs are employed without preliminary tuberculin testing, are relatively inexpensive, and should therefore have a place in mass surveys for pulmonary tuberculosis.—*Va. Med. Mo.*

FACTORS IN ETIOLOGY OF STERILITY

Constitutional—Chronic infection; tonsils; teeth; poor health; anemia; lack of rest; protein starvation; alcoholism; undernutrition; diabetes; T. B.; syphilis; past age of optimum fertility.

Endocrine—Pituitary deficiency; ovarian; thyroid; menstrual disturbance; amenorrhea; oligomenorrhea; hypomenorrhea; polymenorrhea; menorrhagia; anovulatory menstruation.

Faults in Reception—Dyspareunia; vaginismus; frigidity; relaxed perineum; sperm immunity; coitus interruptus.

Hostility to Sperm in Vagina—Excessive vaginal acidity; leucorrhea; trichomona vaginalis.

Cervical Conditions—Abnormal position of cervix; abnormal external os; cervical stricture; lacerations; polyp; acidity of cervical secretions; cervical infection; gonococcal; nonspecific; erosion; viscid mucus plug.

Uterine Conditions—Infantile uterus; uterine fibroids; retroversion; congenital anomalies; endometrial anomalies.

Tubal Occlusion—Unexplained; post-abortional; gonococcal; tuberculous; adhesions subsequent to abdominal operation; angulation of tubes from retroversion; endometriosis; tubal spasm; tubal atonicity.

Defective Ovulation—Endocrine (pituitary, thyroid); peri-ovarian adhesions; thickened tunica albuginia; follicle cystosis; dermoid cysts; persistent corpus luteum.

Husband—(1) Constitutional. (2) Endocrine. (3) Occluded vas. (4) Sperm deficiency.

—*Va. Med. Mo.*

DETERMINATION OF RAPE

The exact time, date and hour of the physician's examination should be carefully noted as well as a careful history of the assault. Permission for the examination must be obtained, either verbally before witnesses, or in writing.

The examination entails: (1) General physical appearance and demeanor. Difficulty in walking is more apt to be noted in minors. (2) Presence or absence of marks of violence on the body, their character and position. (3) Presence or absence of marks or stains on clothing. (4) Condition of the genitalia as to blood; bruising or injury to vulva; presence or absence of hymen; if lacerated, the extent and direction of rupture; presence of menstruation; whether the woman has been accustomed to intercourse. The fact that menstrual blood usually contains epithelial cells, whereas blood from injuries would be free of such cells, must be borne in mind. (5) Presence of venereal disease. (6) Whether pregnant or not. (7) A specimen of the secretion from both vagina and vulva should be studied for spermatazoa.

If the woman is seen early, bruising may not be apparent and it would be advisable to re-examine her some hours later. Slight genital injury, with no evidence of external violence, requires a cautious opinion. Such signs may disappear within four to five days and even severe lacerations may be obliterated in two to three weeks. As stated by Gonzales, "a ruptured hymen would not necessarily signify rape, nor would an intact hymen preclude it." The onset in the woman of a venereal disease of the same variety as that found in the accused constitutes valuable medical testimony.—*West. J. Surg. Ob. and Gyn.*

ACUTE EAR

Treatment of ear infections is a problem for those who assume the responsibility of treating children.

1. Many ear infections will be prevented by early use of nose drops that shrink swollen tissues and by sulfanilamide in treatment of upper respiratory infections.

2. A painful, bulging ear drum in a child with fever that does not subside, should be opened, if there is no response to treatment within 24 hours.

3. Sulfanilamide is advisable and effective when given in graduated doses which become smaller each day. Several courses may be necessary if complications arise.

4. Ear discharge subsides in one-half to one-third the average time when sulfanilamide is used.

5. Mastoiditis is a less frequent complication. When it occurs, medical treatment will usually be effective in most cases, if drainage is maintained through the ear and sulfanilamide is used as directed.

6. Repeated ear infections may be avoided by removal of diseased tonsils and adenoids, treatment of sinus infections and of underlying nasal allergy which is so frequently associated with so-called chronic sinus infections.—*N. W. Med.*

ACCIDENTS

Accidents, preventable or otherwise, constitute one of the greatest and most calamitous risks of existence.

Aside from the psychological element of personal loss, the economic deficit of the *disabling* accident ranks only slightly less than that of the casualty that is a fatality.

Industrial accidents have decreased materially through protective legislation. Unfortunately the preventable accident, according to statistics, indicates material increase.

Even more than illness, accidents are one of the chief causes of physical and economic disability.

Reliable statistics show that annually the population of the United States sustains *ten million disabling accidents*, a daily average of about 30,000, and because of which a consequent body of some 500,000 persons is kept from usual daily tasks by accident. Accident fatalities run about 100,000 deaths per annum.

These figures read like the casualties of war.

Another item that calls for thoughtful deliberation is the fact that low income families ($1,000 per annum or less) and those families on relief has the higher proportion of accidents. Two authorities call attention to the fact that in "relief families" *home accidents* were 43% higher than in families running in the $3,000 income class and upwards.

Of "home accidents" it is reported that 59% are occasioned by falls. Such accidents are due probably to disrepair of the buildings occupied, worn or weakened carpetings or furniture, or the inevitable inconvenient and insufficient lighting arrangements of the establishments of the more destitute.—*Ill. Med. Jo.*

BOOK NOTES

DISEASES OF THE GALLBLADDER AND BILE DUCTS: By Waltman Walters, B.S., M.D., M.S. in Surgery, Sc. D., F.A.C.S., Head of Section in Division of Surgery, The Mayo Clinic; Professor of Surgery, The Mayo Foundation (University of Minnesota); and Albert M. Snell, B.S., M.D., M.S. in Medicine, F.A.C.P., Head of Section in Division of Medicine, The Mayo Clinic; Professor of Medicine, The Mayo Foundation (University of Minnesota). 645 pages with 342 illustrations on 195 figures. Philadelphia and London: W. B. Saunders Company, 1940. Cloth, $10.00.

The authors present the profession with a treatise on diseases of the gallbladder and bile ducts which not only provides interesting reading but is complete, thorough and exact in its composition.

This is a practical medical and surgical review of the experience of the Mayo Clinic over a period of half a century. Theoretical detail is limited to a pleasing degree, only including such material as is essential to the better understanding of the points under discussion. An extensive bibliography appearing at the end of each chapter acknowledges the contributions of others in this field and directs the reader to his articles of choice.

In addition to the authors, other members of the staff of the Mayo Clinic have edited several chapters, including Drs. Jesse L. Bollman, Hugh R. Butt, Howard K. Gray, George M. Higgins, B. R. Kirklin, William C. MacCarty and Thomas B. Magath. Sister Mary William has written the chapter on the technic in the operating room.

Many photographs of specimens, charts and drawings are included.

The differential diagnosis of gallbladder condi-

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*"Treatment of Acute Anterior Urethritis with Silver Picrate," Knight and Shelanski, AMERICAN JOURNAL OF SYPHILIS, GONORRHEA AND VENEREAL DISEASES, Vol. 23, No. 2, pages 201-206, March, 1939.

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tions is stressed in considerable detail. The subject of jaundice is elaborated and the detailed preoperative and post-operative care of patients with this complication is presented. Both authors have long been identified in this field of investigation.

It is with pleasure that one reads the exact treatment as used by the authors with special emphasis on the necessity of the close co-operation of the internist and surgeon in the successful treatment of these conditions. The management of special symptoms and complications and a discussion of the symptoms which follow cholecystectomy and other operations on the gallbladder is ably presented. The chapter on indications for operation attracts our immediate attention.

Exact operative procedure is presented and illustrated by numerous photographs and sketches.

The authors have succeeded in approaching near perfection in presenting this subject, and every member of the profession treating abdominal diseases will profit by a close study of this concise and well-edited volume.—*J. J. G.*

THE PATIENT'S DILEMMA—The quest for Medical Security in America, by Hugh Cabot, M.D.; Reynal and Hitchcock, New Yory; 1940.

The book is written from an idealistic point of view and hence is a valuable contribution to the problems of medical care irrespective of the personal views of the author, which are unobtrusive. The gist of the facts presented are that "good medical care" differs from what has been spoken of as "adequate medical care," and that while much of the general public usually has adequate medical care only a relatively small fraction of the public has the best medical care which scientific medicine affords. There is too great a lag—often years—between the discovery and application of medical facts. To accomplish the purpose of getting the best of medical care to most persons there must be governmental control of medical practice administered by medical experts. All children should have preventive measures against smallpox, diphtheria, typhoid, etc. Parents should see to this; having them do this is a good part of the "dilemma." Horses should have water provided for them and they should be allowed to go to the water and even led to it, but when they will not drink how shall they be made to drink? The author does not tell how to get the American public to fully appreciate the great importance of the application of disease preventive measures, and of prohibiting the work of quacks and the fallacious advertising of quacks and "proprietary medicines." Nor does Dr. Cabot tell how the medical experts might police the medical profession to see that its members are universally up-to-date in their diagnostic and therapeutic measures.

The book is thought provocative and should be carefully and non-prejudicially read generally by physicians and by laymen.—*O. H. B.*

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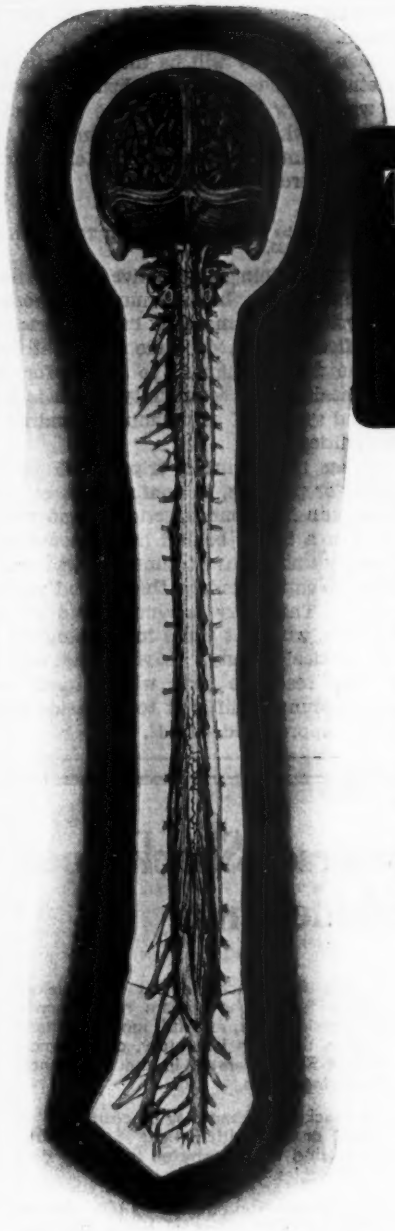

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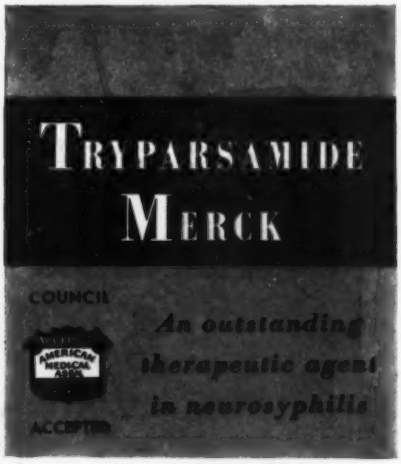

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PICTURE OF HEALTH, by James Clark; illustrated by Guy Rowe; The MacMillan Company; New York; 1940.

This is an interesting small volume of 125 pages of the physiology of the body, told in a unique, intriguing manner. The facts are trustworthy. The work should become a classic or near-classic in this field until that time when many undiscovered truths become commonplace.—*O. H. B.*

LEE ON THE LEVEE (historical novel), by Ralph Cannon; The Saravan House, publishers; New York.

Two famous characters, one a physician, were associated intimately for a time while Beaumont was making St. Louis his permanent headquarters. From 1837 to 1840—one century ago—Lee was sent by the United States government to supervise work designed to maintain the channel of the Mississippi river in *status quo* so that the landings at St. Louis would not become inaccessible to river traffic. At that time St. Louis was a growing settlement and houses were scarce. As a result, Lee and his family were domiciled with Dr. Beaumont and his family in the house known as the Governor William Clark mansion—Clark of Lewis and Clark fame. The intimacy which developed between the families led to a continued correspondence through years after Lee's work in St. Louis was terminated. Lee's letters to Beaumont have fortunately been preserved by Ethan Allen Beaumont, grandson of the great physiologist. These letters obtained from Mr. Beaumont by Dr. Arno B. Luckhardt of the University of Chicago constitute the basis for a most interesting tale woven by the facile pen of Ralph Cannon.

Medical men generally will be interested in the intimate picture of Beaumont and Alexis St. Martin and those who know something of the Mississippi and the boating which took place upon it in the days of Mark Twain and other famous characters, will be doubly interested.—*O. H. B.*

FRONTIER DOCTOR, by Urling C. Coe, M.D.; The MacMillan Company, New York; 1939.

This is an entertaining, instructive, well-written story of the interesting, happy, humorous, sordid, and often physically and mentally hard experiences of a scientifically trained, philosophic, athletic American who devoted himself to caring for the problems of health of a community which is now thought of and spoken of as perhaps a hundred or more communities.

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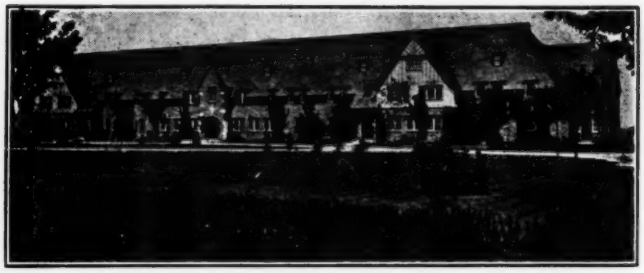

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